

PRINCIPLES OF
DIAGNOSIS AND TREATMENT
IN HEART AFFECTIONS

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PRINCIPLES OF DIAGNOSIS AND TREATMENT IN HEART AFFECTIONS

BY

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PREFACE TO THE SECOND EDITION

THE purpose of this book was to present the recent advances of cardiology in a form that would be understood by the general practitioner, and if possible, to describe the more important phenomena in such a way that they could be recognised by the trained senses of the observer. Such an endeavour was not easy to accomplish, nor can it be said that it was entirely successful, nevertheless, it has helped a great many general practitioners, as the demand for the book has necessitated repeated reprints.

In presenting a second edition I felt that as I had been so long out of touch with general practice, it would be better that it should be edited by one actively engaged in general practice, so I asked Dr. James Orr to undertake this task.

J. M.

PREFACE TO THE FIRST EDITION

THE contents of this book were prepared as lectures to be delivered to the post-graduate students and workers at the Cardiac Department of the London Hospital. The outbreak of the war prevented their delivery, and in publishing them I divide the matter into chapters instead of lectures, while I retain the colloquial form of expression.

A great deal of our recent knowledge of heart conditions has been attained by the use of such mechanical aids as the polygraph and the electrocardiograph. These instruments are not available to the general practitioner, so that if the recognition of diseased conditions were to depend on their use, much of our recently acquired knowledge would be of little practical value. It has been a constant endeavour on my part to recognise the different conditions which these instruments have revealed by employing the ordinary bedside methods of examination, and I am concerned mainly with them in this book. Those who wish for the evidences which the mechanical aids have produced are referred to my book on Diseases of the Heart.

As the main question in every examination of the heart is concerned with heart failure—whether it is present or foreshadowed—one of the objects I have in publishing this book is to present the essential matters connected with heart failure in such a manner that the general practitioner can appreciate them and apply them in his practice.

Another object I have in view is to present this subject in such a form as to lead to a better conception of what clinical medicine means and how clinical investigations should be carried out, and, if possible, to stimulate research on lines which are essential to advance, but which have been wholly neglected.

I have to thank my friends Dr. George A. Sutherland and Dr. R. M. Wilson for their kind assistance and suggestions.

J. M.

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CHAPTER I

HEART FAILURE

The purpose of a patient in consulting a doctor is to find out what bearing his complaint has on his future, and this purpose gives the motive for all clinical research.

A PATIENT consults a doctor because he is conscious that there is something wrong with him. The doctor, in his examination, may detect some sign or sensation which he recognises as a departure from the normal. He may recognise the mechanism by which the symptoms are produced and be interested in it from the physiological or pathological point of view; but these are not the points which are essential to a proper performance of his duties, although they may be contributory to that performance. It is from the patient's standpoint he must view the matter, and, apart from the question of immediate relief, the patient's standpoint is summed up by the question, "What bearing has the cause of this symptom upon my future?"

The necessity of answering this question should be the governing motive in all clinical investigations; for this necessity, if we view it aright, gives to clinical medicine an aim peculiar to itself and distinct from scientific investigation in any other field.

When the patient thinks there is something amiss with his heart, he fears it may fail. It is therefore necessary that the doctor should understand what heart failure is, and the signs by which it is made manifest.

When it happens that the abnormal manifestation indicates that there is something wrong with the heart,

the question may be more clearly realised by asking the patient what he fears. He may not be able to express it, and his ideas may be vague and confused, but what in fact he is afraid of is that his heart may fail. He demands of his physician that he shall tell him whether or not his present symptoms indicate heart failure or foreshadow its occurrence. This is the imperative question which demands an answer in every case with an affection of the heart.

To be able to answer this question it is necessary, in the first instance, to understand what heart failure really is. Heart failure may be defined as *that condition in which the heart is unable to maintain an efficient circulation during the efforts necessary for the daily life of the individual*. This definition is purposely extended to embrace cases of advanced failure as well as those in which the heart failure is just beginning. It is needful to insist upon this point, as the absence of a clear conception of what heart failure means has led to the magnifying of the significance of obvious signs, such as murmurs or irregularities, and to the neglect of the essential symptoms. I make no apology therefore for calling attention, in this respect, to certain features of the circulation which are not usually considered with sufficient care and understanding.

The main elements composing the circulatory system are the blood, the heart, the blood vessels, and the nerves supplying and controlling the heart and blood vessels. The different parts of the circulatory system are so constructed as to facilitate the flow of blood and so to favour the work of the heart. The muscle fibres of the heart supply the force which propels the blood. So far, then, as the efficiency of the circulation depends on the flow of the blood, the muscle force is the only factor concerned, and the inability of the muscle of the heart to supply an efficient force is the direct cause of heart failure. The study of heart failure, therefore, practically resolves itself into an inquiry into

the circumstances which have impaired the functional efficiency of the heart muscle.

Functional properties of the heart muscle are of importance in the maintenance of an efficient circulation.

Before inquiring into the manner in which the heart muscle may fail in its work, it is necessary to consider certain features of the functions of the normal heart muscle. Physiologists have shown that the heart muscle possesses a number of separate functions which are exercised during each beat of the heart. One feature common to all these functions is that exercise of them induces exhaustion, and that, once exhaustion has been produced, the maximum efficiency of function becomes impossible until adequate rest has been obtained. If the heart muscle is stimulated to action before a sufficient rest-period has elapsed, the resultant contraction is always less efficient than the normal. The reason for this is that during the period of activity certain functional changes occur which destroy the accumulated nutritive material, the presence of which is necessary to the exercise of the muscle functions. It takes some time for these materials to be renewed, and the period of what is called "rest" is actually taken up with the restoration of this nutritive material. Hence we may formulate one great law that "a period of rest is necessary after every heart beat." The full significance of this law and its bearing on heart failure will be apparent if we look on the work of the heart as a whole.

When the body is at rest the heart uses only a limited portion of its power. When the body is active the heart calls on its reserve power.

The heart possesses the power not only of maintaining an efficient circulation when the body is at rest,

but also of varying its activity according to the bodily requirements.

We have thus to discriminate between the capacity of the heart to maintain the circulation efficiently when the body is at rest, and its capacity to respond adequately to the needs of the body during its periods of activity.

For convenience of description the former is termed the "rest power" of the heart, and the latter the "reserve power."

When the efficiency of the heart is impaired, the impairment is first indicated by a limitation of the capacity of the heart to adapt itself to the needs of the body during physical exertion. In other words there is a reduction of its reserve power.

When the reserve power is so reduced that practically no effort can be undertaken without distress, the heart may still be able to maintain an efficient circulation when the body is at rest, *i. e.* the rest power is adequate.

As impairment progresses even this becomes reduced and the heart is unable to function adequately even in the most favourable circumstances.

When the rest power becomes inadequate, continuance of the impairment leads eventually to a fatal issue.

Exhaustion of the reserve power may be physiological or pathological, the latter being of the same kind as the former, only occurring with greater facility.

The same principles of exhaustion by effort and restoration by rest, upon which depend the proper functioning of each individual beat of the heart, are applicable in respect of the reserve power. When a healthy individual undertakes some form of severe bodily exertion—for example, running at the "top of his speed"—there comes a time, sooner or later, when he suffers a temporary exhaustion of his reserve cardiac energy. Ultimately, on account of this temporary exhaustion,

he is compelled to desist. If he attempts to run again before a space of time sufficient for complete rest has elapsed, he will not be able to maintain his full speed during so long a period as in the first instance. If he systematically denies himself a sufficient period of rest, his powers of endurance will gradually become more and more restricted.

Now it cannot be too strongly insisted upon that this process, which we recognise as natural in a healthy individual, is identical with the process occurring in an individual having some defect, whether the defect is due to disease of the heart itself or to other factors which embarrass the heart in its work. *The persistent forcing of a heart which is being denied a sufficient period of rest in which to recuperate itself is the real cause of every form of heart failure, no matter what the actual character of the disease may be.* This is the essential fact in the consideration of heart failure. In advanced diseases the amount of work of which the heart is capable may be very limited; nevertheless, danger will not arise unless, and until, too much work is given the weakened heart to do. The term "too much work" must, however, be understood in its true significance. In those advanced cases in which the heart is unable to maintain an efficient circulation when the body is at rest, "too much work" will mean the mere effort of keeping "body and soul together." In a case of this kind, as I have already indicated, the life of the individual is actually in peril.

The mechanism by which exhaustion of the heart is produced and the symptoms which it produces are the same in health and disease.

It will be seen, therefore, that exhaustion of the heart muscle in individuals with enfeebled hearts is of the same nature as exhaustion of the healthy heart; and that heart failure is simply the premature occurrence

of this exhaustion. No doubt exhaustion can be brought about in a greater variety of ways in diseased than in healthy hearts, but this consideration is not germane to the point at issue. Whether it be the action of the heart itself which tends to induce the exhaustion, or the irregular action of the different parts of the heart, or the too great frequency of the rate of contraction, or a progressive lesion, the result is always the same, and the principle governing the production of the symptom is the same; and by recognising this, we get a clue as to the best way in which to prevent the exhaustion or to restore the diminished energy. This principle is equally applicable to conditions of cardiac exhaustion occurring in healthy people. The symptoms are the same in their manifestation and in the mechanism of their production. When I deal with the symptoms of heart failure I shall point out that the only difference between a man in fair health and a man with an affection of the heart which limits his efforts is that, in the case of the latter, certain disagreeable sensations are provoked more readily than in the case of the former. The sensations are identical in kind. Any modifications they may show in different states of health are quantitative only. The patient with a heart affection no doubt tends to suffer a greater degree of exhaustion than the man possessed of a healthy heart. But the mechanism by which the sensations are produced is identical.

The symptoms give no clue to the morbid condition producing the exhaustion.

It will be clear from the foregoing that these sensations produced by exhaustion of the heart muscle, however easily provoked they may be, give no clue to the underlying morbid condition. This fact must be borne in mind. The actual morbid condition present must be determined by the consideration of other evidence

revealed by the history and by the physical examination. The sensations, however much they may vary in degree, are common to all forms of heart impairment. The well-known fact that certain sensations are apt to occur more readily with some than with others, as, for example, pain in aortic disease, in no sense alters this fundamental truth.

When heart failure sets in the earliest manifestation is always a subjective sensation of a disagreeable kind.

It is evident that if the rest power of the heart is unaffected, the individual, when at rest, will present no signs of heart failure. It is also evident that in the early stages of heart failure the signs of exhaustion of the heart muscle will only be found when an effort has been made, as it is only then that the limitations can appear. That there is a limitation can be known in the very early stages only to the individual affected. In other words, the evidence of heart failure in the early stages is entirely subjective; the individual becomes conscious of certain sensations of distress or discomfort on making an effort which, formerly, he was able to make without experiencing these sensations. As the heart failure advances, these sensations are provoked by less effort, until finally the heart is unable to maintain the circulation even when the patient is at rest, and objective evidences such as dropsy and laboured breathing supervene.

To appreciate the meaning of heart failure, we must, as has been indicated, bear in mind that the important factor in maintaining the circulation is the heart muscle. There is a limit to the amount of work which a healthy heart muscle can perform. It is the forcing of the heart to work beyond this limit that produces heart failure. We are justified, therefore, in looking upon heart failure as the expression of an exhausted heart muscle.

Abnormal signs revealed by physical examination must always be considered in relation to the efficiency of the heart muscle.

The principles just enunciated afford a standard for estimating the value of significance of all the signs, normal and abnormal, which may be detected by physical examination. When we meet with an abnormal sign, be it a murmur or a variation in rate or rhythm, or an increase in the size of the heart, we must not, upon the strength of that sign alone, jump to any conclusions. We must realise that that sign does not, of itself, indicate heart failure.

As an outcome of the careful consideration of symptoms and their relation to heart failure, it can be stated that the presence of a sign revealed by physical examination, no matter how abnormal it may seem, is of no serious significance *so long as it is the only sign present*, or so long as there is no limitation of the reserve capacity of the heart. If this dictum be grasped, and the full significance of the principles on which it is based be understood, the practical study of heart affections will be greatly simplified and much clearer and more precise ideas as to the prognosis and treatment of heart affections will be attained. When I come to deal with some of the more common abnormalities, the importance of this statement will be appreciated.

The early signs of the exhaustion of the reserve power are certain sensations experienced by the individual, and produced by exertion.

The symptoms of exhaustion of the reserve power of the heart are not necessarily indications of a diseased or impaired heart muscle. They may be found in healthy individuals, and they only indicate exhaustion for the time being. Actual impairment is, however, indicated when the symptoms are produced with abnormal facility, as, for example, after an effort that the individual was

went to perform in the past without any sign of distress. The point that I wish to emphasise here is that *it is the abnormal facility with which the signs of exhaustion are produced, and not the signs themselves, which is the earliest indication of heart failure.*

The standard for estimating the heart's strength is found by ascertaining the response of the heart to effort.

The standard by which the heart's strength is to be measured is not a fixed standard;—it will vary with each individual examined. The field of normal response to effort varies widely in different healthy individuals. Every man knows the amount of effort he can normally put forth without distress, and this amount of effort is the only practical standard by comparison with which it can be determined whether the field of the heart's response to effort is restricted. It is the appreciation of the fact that in cases of heart failure distress is evoked prematurely that renders it possible to appreciate the true character of any particular case. When there is limitation in the range of effort it is necessary, therefore, to inquire carefully into the history of the case in order to find out when the limitation was first noted, and the circumstances in which it first occurred.

Exhaustion of the rest power is shown by the persistence of certain signs, as dropsy or laboured breathing while the body is at rest.

The sensations which indicate exhaustion of the reserve power persist, and become more readily provoked, as the exhaustion proceeds. By the time that objective signs of heart failure have arisen—*e. g.* dropsy and laboured breathing when at rest or after slight exertion—the heart failure has proceeded a long way. When such objective signs persist while the body is at rest, the reserve energy of the heart is practically exhausted, and the rest capacity is becoming inadequate.

It is well to remember, however—since an exaggerated importance is frequently attached to the absence as well as to the presence of these objective signs of heart failure—that these signs may never appear, even in people who die from gradual and progressing failure of the heart. Thus, I have known physicians give a good prognosis in cases of extreme exhaustion, because, in their own words, “compensation was good,” and there was no objective sign of heart failure. The subjective symptoms of heart failure, on the other hand, are never absent when the heart’s efficiency is in any way affected.

Methods of estimating the cardiac efficiency which do not take into consideration the sensations produced by effort fail to bring out the information essential to a knowledge of the heart’s efficiency.

I am well aware that it may seem surprising that I emphasise so strongly knowledge of such a simple kind—knowledge, indeed, with which the merest tyro is supposed to be familiar. Had I propounded some fanciful blood-pressure scheme, or other means by which one could estimate the heart’s strength according to some mathematical formula, it might have seemed more impressive, if less intelligible, than the comparatively simple method which is afforded by the study of the patient’s response to effort. But no method which fails to take into account the sensations produced by exertion can possibly elicit the information essential for an accurate estimation of the efficiency of the heart.

We are accustomed to regard with amusement the fear and respect with which the backward races treat their idols, yet we are not free from the same tendency ourselves, although the tendency may masquerade under the guise of science. I frequently see eager young men return from a Continental tour, carrying with them some wonderful contrivance—it may be an ingenious stethoscope or phonendoscope, or a curious blood-pressure instrument. They carry the instrument home with

them in the firm belief that they possess a small idol which will reveal the mysteries of Nature. Surrounding all these instrumental methods of investigation there is an element of mystery and a trustful expectation that the fountains of knowledge are about to be unsealed.

I am not decrying the use of these instruments, for they bring to light certain information which enables us to understand better the action of the heart. But a consideration of the essential conditions that lead to heart failure will at once define the limitations of all methods which depend on the use of mechanical contrivances. The chief limitation is due to the fact that the examination of the body, at rest, affords no opportunity of estimating the reserve capacity of the heart.

Another method, that of studying the heart during exercise, by counting the beats before and after some exertion, has been advocated. But here there is a fallacy so great as to render the method misleading. It has been found that in some people with manifestly damaged hearts the rate increases but slightly on exertion, while, on the other hand, in healthy individuals in good training the heart's rate may reach 200 beats per minute on exertion. Measurements have, further, been made of the time which elapses before a heart's rate falls to the normal after exertion, and calculations have been based on certain variations in the blood pressure; but I have little hesitation in saying that none of these methods brings to light the knowledge which is essential.

To gain a fuller insight into this matter, and to understand how completely this aspect of the heart's functions has been ignored by the profession, it is only necessary to turn to the practice of those who may be called the specialists in prognosis—I mean the life insurance examiners. The questions asked in the medical form of an insurance society—which form, remember, represents the accumulated wisdom of bygone generations of examiners—show this very clearly. There are questions

relating to the size of the heart, its rate and rhythm, and the character of its sounds, but there is rarely a single question directed to the elucidation of the most important class of evidence, the evidence which gives the really essential information on which to base a prognosis concerning the patient's future. Some insurance companies have special "heart forms," to be filled up when any abnormal sign is perceived in connection with this organ. I have seen those forms, and, although each of them contained a long series of questions to be answered, not one contained a question designed to elicit what I think I have shown to be the essential information.

It requires careful study and long training before we can interpret the patient's sensations.

One reason for this neglect is the fact that the method we have considered is surrounded by pitfalls into which the unwary are continually falling. There is certainly not one of these phenomena which indicates subjective evidence of heart exhaustion which may not arise from causes other than cardiac, or from conditions of the heart widely dissimilar. Indeed, the confusion in respect of these subjective symptoms is brought before me nearly every day. It is a confusion resulting not only from neglect, but also from misinterpretation. Individuals who have shown some transient sign of exhaustion, who have been breathless or who have fainted, are treated as if they suffered from some affection of the heart. On the other hand, I see, repeatedly, individuals in the greatest danger whose serious condition has never been suspected, because there was present no physical sign indicating heart trouble. In these latter cases, if the patient's reaction to effort, as measured by his own sensations, had been investigated and appreciated, the gravity of the condition would have been recognised.

OBJECTIVE SIGNS OF HEART FAILURE

(Dropsy, enlargement of the liver, congestion of the kidneys, cedema of the lungs.)

As the heart failure advances, the force of the left ventricle, which sends the blood onwards, shows signs of weakening. The effect of this is first seen in the parts where the force of the circulation is normally least—for instance, in the feet and in such organs as the liver and kidney. Hence we get dropsy, enlargement of the liver, and albumin in the urine. The facts concerning these signs are so well known, and these signs indicate so extreme a type of heart failure, that I do not wish to do more than allude to one or two points.

Œdema about the ankles is common in many practically healthy people towards the end of the day, so that, if the heart's efficiency in other respects is good, and we can exclude other causes, such as an interference with the venous flow or conditions such as anæmia, the symptom is of little significance.

I have occasionally been misled as to the cause of dropsy by the coincidental presence of some heart abnormality like a murmur, and have attributed the dropsy to heart failure when it was really due to some other cause. I have seen enlargement of the liver and dropsy treated for years as symptoms of cardiac origin, when in fact the disease was in the liver. I have, therefore, sought for some method of differentiating causes in these cases; so far, I am inclined to think that we never get dropsy or enlargement of the liver without at the same time distinct evidence of dilatation of the heart. Such is the impression I have obtained, but I have not tested the matter in a sufficient number of cases to be dogmatic.

Œdema of the lungs.

In many cases, especially in those cases which are confined to bed—*e. g.* in the fevers—the first sign of heart

failure will be found on examination of the bases of the lungs. The very first signs are the fine crepitations perceived during the first deep inspiration. These crepitations when the failure is beginning disappear with a few deep inspirations, but as the failure proceeds they persist, and by and by impairment of the percussion note at the bases can be perceived; this impairment gradually develops into complete dullness, and at the post-mortem the lungs may be found sodden and engorged.

I have watched this gradual process in a great many conditions, such as chronic heart disease in the elderly who are compelled to lie on their backs as the result of some operation. I have noticed it in fevers, especially typhoid, and have used it as a guide to the extent of the heart's weakness in these conditions. We know that, apart from such an accident as perforation of the bowel, heart failure is the greatest danger in typhoid fever. For many years I have regarded the absence of these symptoms (*i. e.* crepitations and dullness of the bases of the lungs) as of good prognostic significance, and have considered their presence and degree a guide to the gravity of the case. My usual routine method of examination was to start by finding out upon which side the patient usually lay; I then got him to sit up and auscultated the lung base of that side, while he took a deep inspiration. When I detected crepitations I instructed that the patient be raised and at periods during the day be asked to breathe deeply. I reasoned that this sign—crepitation—was due to the failure of the right ventricle to send the blood efficiently through the lungs, and that lying in bed compressed the ribs and interfered with free breathing, while sitting up, on the other hand, freed the ribs. The deep breathing thus assisted the heart in sending the blood through the lungs.

CHAPTER II

BREATHLESSNESS

Breathlessness is produced by stimulation of the respiratory centre in the medulla.

THE respiratory centre is in an ever-varying state of activity. An excess of oxygen in the blood may dull it or may suspend its action for a period; a deficiency of oxygen in the blood, or the accumulation of certain waste products in the blood, may stimulate it to excessive activity. Hence a weak heart that fails at any time to send sufficient aerated blood to the respiratory centre induces breathlessness. It may be that the heart cannot do this when the body is at rest, giving rise then to orthopnœa.

Other factors than the failing heart may over-stimulate the respiratory centre, as certain blood states, the result of the anæmias, diseased kidneys, mechanical conditions that interfere with the aeration of the blood in the lungs, and disease of the lungs themselves.

Breathlessness on exertion.

The most frequent complaint in heart affections is, as we have seen, shortness of breath. This shortness of breath limits its victim's response to effort. Limitation of this kind is not, however, peculiar to cardiac sufferers. Many healthy people are short-winded—that is, they have so little reserve capacity that it is speedily exhausted; others can undertake an extraordinary amount of effort without any distress. In view of this large individual variation, it is obviously impossible to fix an absolute standard of capacity for effort. The comparison must always be made with the

previous capacity of the individual under observation. Thus, an individual who has been accustomed to take a considerable amount of exercise discovers suddenly a limitation of his powers. This may be very slight and may not at first attract much notice. Nevertheless, the limitation may indicate cardiac inefficiency. We have seen that the amount of reserve energy available varies in healthy people. Bearing this fact in mind, we shall not experience great difficulty in discovering by means of a careful appreciation of the patient's sensations whether the limitation is normal or is due to impairment.

This breathlessness, which arises as the result of effort, is a symptom common to all forms of heart exhaustion, slight and temporary as well as serious. But it occurs also in other conditions—*e. g.* lung affections and wasting diseases—so that no conclusion as to its real nature can be drawn until other phenomena have been investigated. A weighing of the resulting evidence will reveal which of the many provocative causes is at work in the case of the particular individual under consideration.

Breathlessness at rest (*orthopnoea*).

When the breathing is laboured while the patient is at rest, so that he is compelled to assume a sitting posture, the heart's impairment is extreme. But before deciding that the distressed breathing is cardiac in origin, we must exclude the various forms of respiratory disease which induce such extreme breathlessness—*e. g.* asthma, pleurisy, pneumonia, emphysema, and intrathoracic growth. When due to enfeeblement of the heart, the distressed breathing is generally the outcome of a long period of persistent over-exertion. The real cause of the trouble is thus insufficient rest. In these cases the history may reveal the fact that the onset was gradual and extended over months or even years. Rest in bed will often of itself restore a certain amount of strength to the heart and cause the breathlessness to disappear gradually. When the breathlessness occurs rather

suddenly, the likelihood is that it has been caused by the heart taking on an abnormal rhythm—for example, auricular fibrillation—in which the ventricle is stimulated to rapid and irregular action. Extreme breathlessness may set in within a few hours after the onset of auricular fibrillation, though usually it does not appear for a few days or weeks. Its onset in such cases is precipitated by the individual attempting to do his ordinary work while his heart is hampered by the abnormal rhythm. The ease with which breathlessness is induced and the response made by the heart to treatment of the condition give us the data on which we can estimate the functional efficiency of the heart. Persistence of this form of dyspnœa (dyspnœa due to auricular fibrillation) is often associated with dropsy, enlargement of the liver, and a tendency to lividity of the face and extremities.

Cheyne-Stokes breathing.

There is a type of respiration in which the breathing ceases for a short period (10–40 seconds), and then gradually begins again; from the period of cessation the breathing increases in intensity till a maximum is reached, gradually subsiding until it again ceases. Sometimes these phases are very marked; at other times they are so slight that they cannot be detected unless the patient is watched during his sleep. This breathing—known as Cheyne-Stokes—may occur when the patient is sitting quietly in his chair; usually, however, it only occurs when he is asleep or dropping off to sleep. Sometimes the patient passes into sleep during the apnœic period and may resume a conversation when the breathing becomes re-established. Or he may continue to talk, but in a faint voice, during the apnœic phase and return to his louder voice during the respiratory phase.

Although Cheyne-Stokes breathing is not usually discovered as a subjective sensation, on account of the fact

that many of those affected by it are quite unconscious of its presence, yet for convenience of description it may be included here. Its presence may be suspected when the patient describes suffocating sensations, which keep recurring, or states that he awakes feeling suffocated. This sense of suffocation is usually due to the recurrence of the apnœic phase; sometimes it is so distressing that the patient will suddenly spring out of bed in an extremity of terror.

Cheyne-Stokes respiration has a varied clinical significance. Many elderly people with no particular disablement, beyond the considerable enfeeblement so common in advanced years, show it. In people with high blood pressure, advanced sclerosis of the heart and arteries, it is generally a sign of approaching dissolution. I have seen it in several cases of extreme heart failure, with auricular fibrillation and auricular flutter; and in these cases it has quite disappeared when the heart failure was relieved by digitalis.

Cardiac asthma.

There is a form of dyspnœa which comes on suddenly with great severity. It is so often associated with marked changes in the heart that it has received the name of "cardiac asthma." It occurs most frequently in the night. The patient goes to bed and may fall asleep for a few hours; he is then awakened by a sensation of suffocation, which forces him to sit up. He breathes heavily and struggles to get into a position which will allow him to breathe with freedom. This dyspnœa may last for half an hour or longer, and may then gradually subside, so that the patient is able to go off to sleep again. Usually, however, he remains propped up in bed, and if he sleeps his sleep is troubled and disturbed, with a fear of the recurrence of the distress. The attacks resemble ordinary asthma and may be quickly relieved by a hypodermic injection of morphia.

This condition occurs most frequently in elderly people with degenerated heart muscle, high blood pressure, and changes in the arteries. These conditions may also, it should be noted, occur in middle life, and may be associated with albuminuria.

Cardiac asthma is a very grave sign, especially when it is associated with *pulsus alternans* or *angina pectoris*, and is usually a prelude of death, so that the duration of the individual's life can be reckoned by months; in milder cases, on the other hand, where the heart responds to treatment, life may be prolonged for a few years.

For some years now I have been inquiring into the relation of this condition to Cheyne-Stokes respiration, and I find that the latter condition is present at times with cardiac asthma. I find again that in most cases the patient wakes in distress during the apnœic stage, and the attack of severe breathlessness which then develops is identical with what is called cardiac asthma. I was in consequence disposed to look upon Cheyne-Stokes respiration as the origin of the attacks of cardiac asthma in all cases; but Sir T. Lewis notes that, while he was examining a patient, the latter suddenly developed an attack, with no preliminary Cheyne-Stokes breathing.

CHAPTER III

SYNCOPE

Syncopal attacks or fainting, in which the patient becomes limp and unconscious, are due to a deficient supply of blood to the brain.

SYNCOPE may be due to loss of blood, to the failure of the heart to send a sufficient supply of blood to the brain, to the heart itself being the subject of some abnormal action, or being depressed in its action as the result of vagal stimulation, or it may be due to the collecting of the blood into the large abdominal veins and the veins of the periphery, with consequent changes in the cerebral circulation.

Syncope from venous stasis.

Taking the last condition first, we find it the most common form of syncope. Many young people faint when in a close atmosphere—*e. g.* schoolboys attending chapel; many women of anæmic, flabby habit faint under similar circumstances; soldiers on the march, especially in hot weather, faint, probably because their heavy clothing keeps their bodies so warm that the peripheral vessels are dilated to an extent sufficient to deplete the brain. In some people the tendency of the blood to drain into the large abdominal veins is so marked that the fainting attacks are easily produced. (See Chapter VI.)

Syncope from vagal stimulation.

When an impression is made by means of a violent mental stimulus—for example, alarming information or

the sight of some harrowing scene—syncope may follow; again, violent intestinal peristalsis, or a powerful afferent stimulus, as, for example, that attending the opening of an abscess, may produce syncope. The pulse in such cases may become infrequent and of very low tension. The cause of the depression here is no doubt vagal in origin.

There are two kinds of cardiac action which tend to induce loss of consciousness—that in which the ventricles stand still (*e. g.* heart-block), and that in which they beat at such a fast rate that the output is greatly reduced (*e. g.* auricular flutter).

The mechanism of heart-block.

In heart-block the attack of unconsciousness occurs so suddenly that the physician has seldom the opportunity of recognising all the features present. In rare cases of heart-block, when the attacks occur frequently, he may get an opportunity of detecting some of the more prominent features. The condition called heart-block is due to the stimulus of contraction, which arises in the auricles, failing to pass along the bundle of fibres that joins the auricles to the ventricles (see Fig. 1). The result of this failure is that the ventricles may beat at an infrequent rate. It will be readily understood that as disease is progressive there are different effects produced according to the stage reached by the disease. The first effect of disease of the auriculo-ventricular bundle is a delay in the transmission of the stimulus from auricle to ventricle, due to the fact that the damaged fibres do not speedily recover from exhaustion after being exercised.

This delay cannot be recognised without instrumental aid, except in those rare instances where there is mitral stenosis, when a pause occurs between the pre-systolic or auricular systolic murmur and the first sound, causing what is described as a mid-diastolic murmur. When the auriculo-ventricular bundle of fibres becomes

more damaged, the recovery of the fibres, after being stimulated, is further delayed, and the ventricle at times

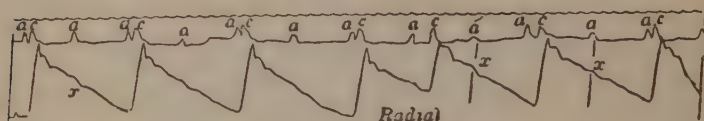


FIG. 1.

Slow, irregular pulse due to partial heart-block after an attack of influenza. The upper tracing is from the neck, and was taken at the same time as the radial tracing. The waves *a* are from the jugular vein and are due to the contraction of the auricle, and the waves *c* are due to the carotid pulse, and these *c* waves correspond exactly in time to the radial beats. It will be noted that there are two auricular waves (*a*) to one carotid (*c*) or radial beat, except in one instance, where the *c* wave and the radial beat appear earlier. Note that the *c* wave follows immediately on the *a* wave, except in the instance when the *c* wave appears earlier, and there is a distinct interval between *a* and *c*. The explanation is that there is an impairment of the bundle connecting the auricles and ventricles, and after the passage of the stimulus from the auricle to the ventricle the power of conducting the stimulus is so exhausted that it fails to respond to every second ventricular contraction, excepting in the one instance where there is a response, and where the interval between *a* and *c* is lengthened, showing a delay in the transmission of the stimulus. (See Fig. 2.)

fails to respond to each auricular contraction, so that ventricular beats drop out. This may happen at rare



FIG. 2.

Diagram of Fig. 1. The downstrokes in the space *A*, represent the contraction of the auricle, the downstrokes in the space *V*, represent the contraction of the ventricle, the slanting lines in the space *a-c* represent the passage of the stimulus from auricle to ventricle (the *a-c* interval). The diagram shows that the ventricle responds only to every second auricular beat, except in one instance, when the increased length of the slanting line represents the prolonged *a-c* interval in Fig. 1.

intervals at first, but with advancing disease of the fibres it gets more frequent (Figs. 1 and 2). A stage is at last reached when the fibres fail, for longer or shorter

periods, to convey a stimulus (Fig. 3). When the fibres no longer convey a stimulus between auricle and ventricle, the ventricle may stand still for a brief period, then take on an independent rhythm, or the normal rhythm may be resumed. When the ventricle takes on an independent rhythm, it may beat in a manner bearing no relation to the auricular contractions.

The Stokes-Adams syndrome.

In most cases where the ventricle takes up this independent action it will go on for an indefinite number of years, beating regularly about thirty times per minute. But, before it settles down to this rate, there are periods alternating with the periods of complete heart-block when the stimulus does still pass from auricle to ventricle. It is at this stage of the disease, when the stimulus first fails to pass from the auricle to

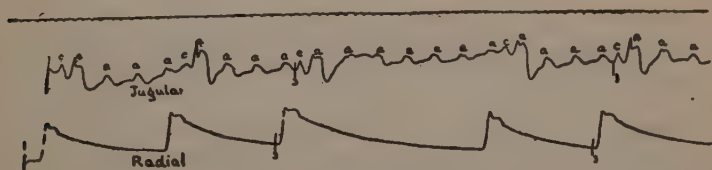


FIG. 3.

The long pause in the radial tracing is due to partial heart-block with a prolonged standstill of the ventricle. In the upper tracing the waves *a* are due to the auricle. This tracing was taken two days before the patient's first attack of loss of consciousness.

the ventricle, that the phenomenon of Stokes-Adams syndrome is most common. At this period there is often a little delay before the ventricle takes up its independent rhythm, and, therefore, the ventricle may stand still for a longer or shorter period. During this pause no blood reaches the brain and the individual becomes unconscious; and hence there arises the train of phenomena described under the name of Stokes-Adams syndrome—loss of consciousness with convulsions.

In one case, from which I took continuous records extending over an hour, the patient had, in the hour, thirty attacks of loss of consciousness. When conscious he would talk quietly and rationally, then his voice would trail off to a mumble and he would become unconscious. He would recover and resume the conversation where he left off. The record showed that before he ceased to talk the left ventricle had ceased to beat; it was ten seconds after the beating ceased that he completely lost consciousness (Fig. 4). Usually the ventricle started to beat after stopping fifteen to sixteen seconds. If it stopped sixteen seconds the patient became convulsed with slight twitching of the muscles of the face and arms. If the stoppage was prolonged to twenty seconds there was a strong convulsion of the whole body. In the attack, accompanied by slight convulsions of the face, he felt the twitching of the muscles as he recovered. I cite this case because it gives a clear conception of the

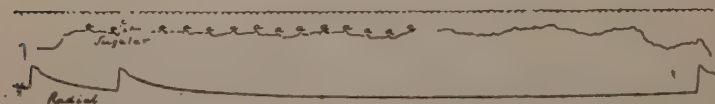


FIG. 4.

The upper tracing is from the jugular vein, and shows small waves, *a*, due to the auricle. The radial tracing after two beats shows a long pause due to standstill of the ventricles, lasting seventeen seconds. During the pause the patient lost consciousness, which returned as soon as the ventricles began to beat. It will be seen that the auricle contracted at a rate of 100 per minute during the standstill of the ventricles.

agency by which syncope and convulsions are produced, and the time it takes for complete cessation of ventricular output to produce these phenomena.

Attacks of syncope are not infrequent in cases of auricular fibrillation, due also to a peculiar form of heart-block. There are rare cases where auricular fibrillation occurs with complete heart-block, and syncopal and epileptiform seizures may occur.

Syncopal attacks due to heart-block.

It happens not infrequently that the account of the attacks of loss of consciousness is dependent on the patient's own description, and he can often describe his experience so clearly that the nature of the attack can be surmised with fair certainty. When a patient states that he loses consciousness for a brief moment—a few seconds—and immediately recovers—sometimes falling down, it may be surmised that his attack is due either to a temporary heart-block or petit mal. If the patient is young the latter may be suspected. If in later life—over forty—the former. In the case of heart-block a graphic or electro-graphic record may reveal a delay between the contraction of the auricle and ventricle, but even without this delay the surmise is fairly certain; I have seen several such cases ultimately develop complete heart-block.

I shall not again refer to heart-block, because, after all, it is a rare condition, but before leaving the subject I may say that, in treatment of this condition, we are unable to influence the rate of the ventricle. We tried in the hospital all sorts of remedies without success. Many patients recover spontaneously from the tendency to attacks of unconsciousness, and these attacks disappear when the independent ventricular rhythm becomes established. The independent rate is usually about thirty per minute. Other patients die in our presence and we are incapable of giving them any help.

Syncope from extreme frequency of the heart rate.

The deficient supply of blood to the brain, which induces syncope, may arise from the ventricle beating so frequently that its output becomes greatly diminished. There is a condition where the auricles beat at a rate sometimes as high as 300 beats per minute and over. To this condition MacWilliam has given the name of

auricular flutter. In the majority of cases the ventricle does not respond to every auricular beat. When, however, the ventricle responds to every beat, the patient is in great distress, and feels as if he is about to fall and lose consciousness. Sometimes he does fall,

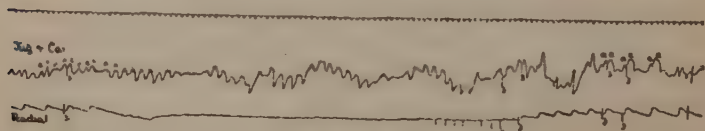


FIG. 5.

Tracings taken during a short attack of auricular flutter, during which the patient lost consciousness. From the character of the radial tracing it can be inferred that the ventricle contracted at the rate of 200, but the output was so small as barely to affect the radial pulse. (Theodore Thompson.)

and the unconsciousness may last for a few minutes or for a few hours, the pulse at the wrist being imperceptible or barely perceptible (Fig. 5). In one of my cases unconsciousness lasted for four hours, the patient scarcely breathing; the doctor in charge thought several times that the man was dead. The patient recovered and lived for ten years, the attacks occasionally recurring (see Chapter XIV).

CHAPTER IV

ANGINA PECTORIS

The pain of angina pectoris can best, for practical purposes, be considered as an expression of exhaustion of the heart muscle, generally from insufficient blood supply.

So far we have been dealing with the signs of heart failure which are due to functional inefficiency, occasioned by the failure of the heart to send an adequate supply of blood to the other organs of the body.

We have now to consider another type of heart failure, the symptoms of which differ from those already described in that they do not depend upon diminished ventricular output.

In angina pectoris the efficiency of the heart, as measured by ventricular output, is not materially interfered with.

The symptoms of this condition do not arise from inadequate blood supply to the various organs of the body, but are referable to exhaustion of the cardiac muscle, generally from the insufficiency of its own blood supply, and are directly attributable to the heart itself. This is manifested by a well-defined syndrome, the most characteristic symptom of which is pain of a distinctive kind.

From a study of the facts the following conclusions seem warranted, and are put forward not only because they are supported by convincing evidence, but because they seem reasonable, and present the matter in such a form as to be of real use in estimating the symptomatic value of this pain from the points of view of prognosis and treatment.

The peculiar regions in which the pain is felt indicate its cardiac origin, while its distribution and accompanying phenomena indicate that the stimulus passes into the central nervous system.

The pain of angina pectoris is manifestly due to a stimulation of certain regions in the central nervous system. The places where the pain is first felt, the areas into which it radiates, and the appearance of areas of hyperalgesia of the skin and deeper tissues, all point to a central stimulation. The region affected in the majority of cases is shown in the shaded area of Fig. 6. It will be seen that the pain extends in a somewhat peculiar manner over the left side of the chest into the inner side of the left arm, forearm, and hand. The pain may start in any part of this region, and may radiate to any other part. Most frequently it appears within the shaded area on the chest and radiates to the arm; on the other hand, it may, on rare occasions, start in the forearm and remain there, displaying great severity; again, it may start in the forearm and radiate into the chest and be severe there. It may also be found in similar regions on the right side, and in the neck and along the jaw.

In addition to pain other signs may be discovered. These include contraction of the intercostal muscles, giving rise to a feeling of severe constriction of the chest, and increased flow of saliva and of urine; they point, all of them, to a stimulation of the central nervous system.

The pain is frequently associated with effort. In most cases the attacks of pain are provoked by effort or excitement. When provoked by effort, they generally cease immediately after the effort is stopped. The pain may be slight at first and may pass off as soon as the effort ceases, but if the effort be persisted in it may increase rapidly and become so violent that the patient is compelled to desist. In addition to the pain, there may be a sense of constriction, as if the chest were being held in a vice; this sensation corresponds

to that encountered in pleurodynia. There may also be a sense of suffocation and a feeling of impending death.

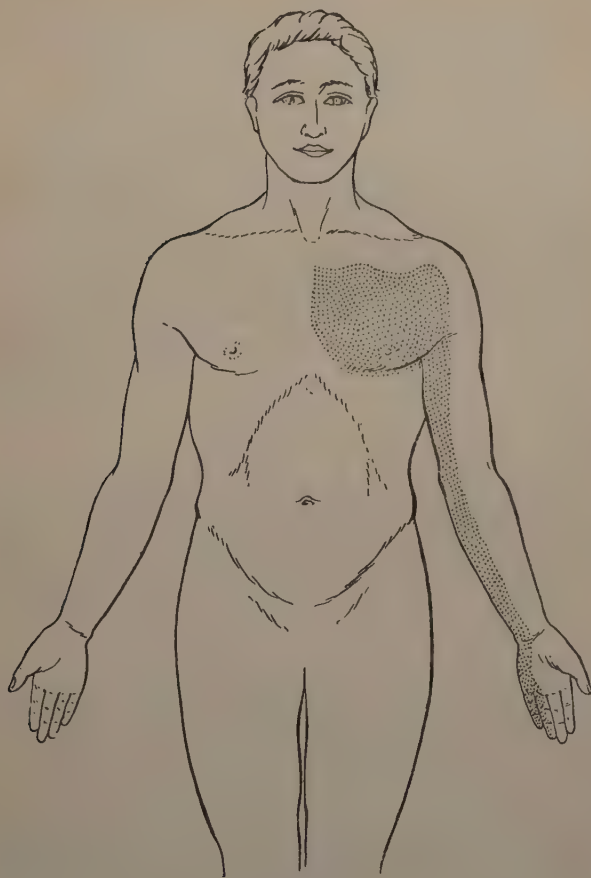


FIG. 6.

The shaded area shows the characteristic distribution of pain of cardiac origin. The skin of the area may be found in a state of hyperalgesia after an attack of angina pectoris. The pain may not be felt in the whole of this region, but only in portions—sometimes limited to the chest, on rare occasions limited to some part in the arm. The hyperalgesia also may have but a limited distribution.

While effort is the most common exciting cause of the attack, and while the attack in the majority of

cases comes on while effort is being made, it happens sometimes that the onset of pain is delayed. The attack may not occur until some hours after the effort has been made. Again, there are cases in which the pain appears where the heart has been exposed to no particular effort, but in which there is a history of a long period of over-exertion preceding the onset of the angina. It is well to bear in mind that pain may be produced by over-exertion in healthy as well as in diseased hearts.

To appreciate this view of the symptoms produced by exhaustion of the heart muscle, it is necessary to consider the circumstances in which pain is most frequently produced. We can accomplish this by observing what happens in the case of the healthy young when over-exerted and by comparing their state with that of men passing into old age who have also been over-exerted.

If we take a number of healthy youths and get them to run as far as they can, each of them will at last slacken speed because of some sensation of distress. The majority will suffer from dyspnœa, a few will suffer from a disagreeable sense of constriction across some part of the front of the chest, or over the sternum at its upper part.

In the young recovering from an illness excessive exertion will occasionally induce actual pain, which will be referred to the left chest; while, in rare instances, healthy young people suffer from this pain after violent exertion.

A number of elderly individuals who have had attacks of angina pectoris have told me that, before the pain appears, they experience a sense of tightness across the chest. They describe this sensation as being exactly like the feeling which stopped them when running in boyhood. This sense of constriction corresponds with the "vice-like grip" of which patients complain during attacks of angina pectoris.

The most frequent physical conditions provoking angina pectoris are the degenerative changes which accompany advancing years.

Many men between fifty and sixty years of age, who are actively engaged in business, are seized with all the typical signs of angina pectoris. Considering how anxiously this condition is viewed, it is necessary for the physician to form an opinion as to its gravity. To do this intelligently it is necessary to bear in mind certain essential facts.

In everyone, as the years pass, changes of a degenerative nature occur. Fibrotic changes in the heart muscle render it less efficient, while changes in the cardiac arteries interfere with the flow of blood to the same structure. Exhaustion of the heart muscle gradually supervenes. Notwithstanding this, the individual may lead a strenuous life till he is pulled up by pain. Atheroma of the coronary artery is a condition frequently met with in people who suffer from angina pectoris; and the part it plays seems to be two-fold; first, in many cases the smaller arteries are obliterated, and, secondly, the changes in the artery wall interfere with its power to dilate. Starling has calculated that the supply of blood to the heart muscle increases three-fold during the period of activity; to meet the demand for more blood the arteries dilate. The degenerated arteries, on account of their rigid walls, are unable to respond to the call for more blood, and, as a consequence, exhaustion of the muscle is provoked. This is analogous to the pain on effort produced by an atheromatous condition of the arteries in the legs. Many elderly people who can walk for miles at a slow rate are compelled to stop if they attempt to hurry, or run, by the intense aching which occurs in their feet and legs.

It is not difficult to understand that hearts with rigid arteries are bound to afford evidence of inefficiency, and, on inquiry, such evidence can generally be elicited. If closely interrogated, patients will invariably acknow-

ledge that, some time previously, it may be as much as five or ten years, they were conscious on occasion of sensations of a disagreeable kind on exertion, such as breathlessness and a sense of constriction across the chest. They ignored these sensations and continued their work. These sensations indicated that, at the time when they occurred, the periods of rest were not sufficient to restore the exhausted muscle, and that, even then, some slight changes were going on which impaired the efficiency of the muscle. In spite of this impairment, the individual continued to work as hard as when he was in the flush and vigour of manhood. Exhaustion, being in the first instance ignored, became finally so extreme that it culminated in an attack of angina pectoris.

We are able to recognise, therefore, that pain and distress result not merely from the degenerative changes that accompany advancing years, but also from the fact that too often an impaired heart is forced to work as hard as it did before any changes occurred in it. The presence of impairment and refusal to recognise that it is present mean that the periods of rest accorded to the heart are not sufficient to permit of restoration of its reserve capacity. The fundamental importance of inquiring into the life of the patient will now be evident. If the inquiry is conducted systematically it will be found that the condition that provokes the exhaustion is over-work—which is not necessarily excessive work, but is always work of a more strenuous character than the degenerated heart can undertake. We may place in this category such troubles as worry and sleeplessness—in other words, circumstances which interfere with the period of rest necessary to restore the exhausted reserve energy. In some cases the heart muscle may be injuriously affected also by toxic agents, such as tobacco, alcohol, and the toxins of infective organisms.

Angina pectoris affords an indication of the extent of impairment of the heart muscle.

As the occurrence of angina pectoris indicates some damage to the heart muscle, it is necessary, in each case, to form an estimate of the extent of this damage. This can be accomplished with considerable accuracy :

(1) By finding out the amount of effort the individual can make under the most favourable circumstances.

(2) By noting the effect of treatment.

Many people afflicted with angina pectoris are unable, starting from rest, to walk more than fifty or a hundred yards without being distressed. This distress may arise during several subsequent attempts, and then may pass off, five or ten miles being accomplished without distress. This remarkable fact indicates that in these hearts a good deal of healthy muscle is present and that the attacks of distress are provoked by the presence of some factor which is not myocardial weakness—for example, a full stomach. (In many cases the pain is provoked only during exertion indulged in after a meal.) The ability of individuals to undertake considerable effort at one time, and their inability to undertake effort at another, afford a very useful means of estimating the amount of efficient heart muscle. It may, for example, be taken for granted that when a man is able to undertake considerable effort without discomfort he retains a good deal of healthy muscle. The fact that occasionally slight effort provokes pain in his case does not alter this conclusion. It will be found that, at the times when he is restricted in his activity, some secondary influence is at work the removal of which enables the heart to regain a measure of strength. The limitations of effort must therefore be ascribed to this secondary influence, and not to the condition of the heart muscle. Again, the extent of myocardial damage can be estimated by noting the effect of treatment. In

all cases in which effort is the provoking cause of angina pectoris, it may be taken for granted that, during a long period of time, the heart has been forced to work above the measure of its efficient capacity, and that therefore the muscle has been exhausted. A period of rest or of liberation from the provoking causes will enable the heart to regain some of its strength, and this added strength will be recognised by an improved response to effort, from which the extent of damage to the heart can be estimated.

In people who suffer from angina pectoris the liability to attacks varies markedly at different times. This is a common experience. For example, during many weeks or months the patient may be comparatively free from his trouble, and may be able to undertake considerable effort without distress, while at other times his attacks are readily provoked. It is impossible to account in every case for this variability. Periods of sleeplessness or of worry may be the provoking cause in some instances, while in others digestive disturbances appear to have an influence in inducing the liability to attack. Systematic over-exertion may also provoke the tendency; and again it must be pointed out that this over-exertion need not be great exertion; over-exertion in this sense is only exertion greater than the heart can efficiently undertake. If the means, referred to in books, by which relief or cure can be brought about are considered, it will be found that a period of rest is always included among them, and this in spite of the fact that the physician may attribute his success to some special treatment, such as a drug, a diet, or a bath.

The description just given applies to the vast majority of cases of angina pectoris. It will be seen that, regarded from the point of view we have adopted, angina pectoris is but the expression of exhaustion, and is not, therefore, necessarily a serious condition in the usual meaning of that term—*i. e.*, that death is impending.

The changes which it indicates, irremediable changes

which impair the heart's efficiency, and which are slowly progressive, are, after all, only those changes which may be expected to occur with advancing years.

Angina pectoris may indicate grave progressive disease.

There are, however, cases in which angina pectoris develops with great severity and ends speedily in death. On the whole, these cases are rare. When they do occur, it will be found that the changes inducing the exhaustion (aortic disease, disease of the coronary artery or of the myocardium) have advanced to an extreme extent before the appearance of the pain. When the changes are of the senile degenerative type, there is always a history of cardiac inefficiency before the onset of pain—as revealed by breathlessness on exertion. The attacks of pain are easily provoked, and treatment avails little. When, therefore, we find that slight exertion invariably provokes pain, and that little or no improvement takes place on rest, we must look upon the condition as a grave one. Another grave form of angina pectoris is found in cases of syphilitic aortitis. This disease may progress with great rapidity, and the earliest symptom may be pain, sometimes of a severe kind, referred to some part of the area shaded in Fig. 6, page 29. When pain of this type occurs in the young or middle-aged, inquiry should be directed to the determination of the presence of syphilitic infection. If these syphilitic cases be watched, an aortic diastolic murmur may be observed to make its appearance. But even in cases in which no murmur appears, the increasing ease by which the pain is provoked should always raise the suspicion of syphilitic aortitis.

In one of my cases in which we could detect no physical abnormality the pain was provoked by the slightest exertion, and the patient suddenly died a few months after the first appearance of the pain. At the post-mortem examination we found in the aorta, above the valves, a great thickening of the internal lining

membrane, which obstructed the mouth of the left coronary artery; in the wall of the left ventricle the muscle fibres were breaking down because of the lack of the blood supply.

In a few cases I have seen apparently good results from energetic anti-syphilitic treatment, but in the majority little benefit has resulted.

Attacks of angina pectoris when the patient is at rest imply extreme exhaustion of, or active changes in, or poisoning of, the heart muscle.

In the vast majority of cases the pain comes on in response to effort, at the time effort is made or shortly after. When the degeneration which causes the exhaustion of the heart is extreme, attacks of pain occur during sleep or when the patient is at rest. In these cases the pain is also provoked easily by exertion. There are, however, cases in which the pain does not appear in response to effort, but only when the patient is at rest; these people can sometimes undertake a fair amount of effort without suffering pain. The majority of the cases of this type depend upon some toxic condition affecting the heart muscle. This toxic condition may be of intestinal origin or may be the result of infection or excessive indulgence in tobacco. Further reference will be made to these cases in connection with the hyper-sensitive nervous system and the poisoned heart. In a few cases I have seen pain associated with subacute rheumatism start in this way. The subacute rheumatism is probably due to a condition resembling the fibrositis of the skeletal muscles. These cases have all recovered. In one case in which pain came on only when the patient was at rest, some obscure infection finally supervened and caused pericarditis, which was followed by death; I have been unable to account for the condition.

The view of angina pectoris as an expression of exhaustion of the heart muscle affords a conception which gives a basis for a sound prognosis.

In the descriptions I have given I have dealt with those people who form the great majority of cases of angina pectoris. From these descriptions it will be gathered that angina pectoris is but a sign of the change that accompanies advancing years. It follows, therefore, that the outlook is, in the majority of people, favourable, particularly when there is present no complicating affection, such as chronic Bright's disease, or marked arterial degeneration.

If the significance of the anginous pain as an expression of exhaustion be recognised and the individual affected by it be able to lead a life at a lower level, so that attacks can, to a great extent, be avoided, the individual may look forward to reaching an advanced age, so far as his heart is concerned. I have watched many people, who have had to lessen their efforts because of angina pectoris, lead useful lives at a lower level during ten, fifteen, and even twenty years.

When, in spite of an easing of the routine of the daily life, the anginous pain is provoked by slight effort, or occurs at times when the body is at rest, the outlook is grave. This is the case especially in men. In women, in whom generally the hypersensitive nervous system plays an important part, the outlook is much less grave. In them less grave lesions of the heart may cause long-continued suffering. In some aortic cases, too, pain is easily induced, long before there is any actual danger.

Some people have a nervous system which is abnormally sensitive to stimulation.

In the production of pain and other sensations dependent upon heart trouble, there are two main factors concerned—the heart and the central nervous system.

In describing angina pectoris I have so far referred chiefly to the damaged heart. We know that many people with damaged hearts suffer no pain, so that, to account for the pain, we are bound to conclude that a partial explanation is furnished by the variability in the sensitiveness to stimulation of the central nervous system. This system is not equally susceptible in all people. There are people, however, with a nervous system so sensitive that pain is produced with great facility. This hypersensitive condition may be natural or it may be acquired. Thus, abnormal sensory phenomena, such as pain and hyperalgesia of the skin, are more frequently met with in women than in men. This tendency to abnormal sensory phenomena is greatly increased by any form of illness of an exhausting nature, and by a life of worry and overwork. Hence we find pain and tenderness in the left chest in patients affected by a great diversity of conditions, ranging from grave disease of the heart to slight functional weakness.

The hyperalgesia, as shown by tenderness on pressure of the skin or muscles of the left chest and of the left breast, indicates a lowering of the threshold of stimulation in certain areas of the central nervous system. In certain circumstances, a stimulus reaching this region, from sources other than the heart, may produce a typical attack of angina pectoris. In one patient, scratching of the skin under the left breast, and even the pressure of the stethoscope, produced an attack. Another patient, near unto death and who died in an attack of angina pectoris, dared not move his left arm because such movement often induced an attack. In cases of slight functional weakness of the heart, with an excessively sensitive nervous system, we may get attacks of pain of the greatest severity. In order to determine whether or not the cardiac pain is of a serious nature, it is necessary to review the whole history of the case, to consider not only the state of the heart, but also the state of the nervous system, and particularly to find out

whether or not the patient is normally of a neurotic temperament. If it be determined that there is that indefinite condition, which I can only describe as a hypersensitive nervous system, but which is easy to recognise, then search must be made for the cause which has induced this condition. You will find the condition most typically shown in people, especially women, who have had long periods of anxiety and worry, with little and interrupted sleep—for example, the mother of a family in which there has been much sickness, or a daughter who has had to work hard in the day and has to attend her ailing parent at night. Again, diseases of various kinds, especially those causing great exhaustion—*e. g.*, loss of blood—may be the provoking cause. Infection may also be an important causative factor; the most severe case of suffering of this kind I ever saw was a woman with a pelvic abscess. An attack of pain of the most agonising description was provoked by the slightest effort, and recovery only took place when the abscess was evacuated.

In these people there are often evident other phenomena, usually of a vaso-motor origin, such as a tendency to flushing, and, at other periods, to coldness of the hands. At times the attacks of pain are accompanied by feelings of extreme cold and rigor-like shivering. The shivering attacks may be independent of pain. The patient to whom I have just referred, who suffered from severe attacks of pain, again became ill a year after her recovery, and had numerous attacks of a rigor-like kind without pain. These attacks disappeared after a discharge of pus into the bladder.

In milder cases there may be no attacks of pain, but simply aching in the left chest and arm, or tenderness on pressure of the left breast and surrounding tissue. Such phenomena, often accompanied by no physical sign pointing to disease or weakness of the heart, are nevertheless an expression of some exhaustion of the heart muscle, and often you will find that they disappear with

the improvement in the patient's strength and return with exhaustion.

The recognition of the hypersensitive nervous system is essential to diagnosis and prognosis in angina pectoris.

It will thus be seen that cases of angina pectoris fall into one or other of two great groups.

In the first group, that of organic or primary angina, the symptoms are referable to organic changes in the heart or blood vessels, *e. g.* the coronary arteries, and are of grave import. In the second group, that of secondary angina, the symptoms depend upon the increased sensitiveness of the central nervous system to various stimuli, and are not associated with any liability to heart failure.

The prognostic significance of these cases is that of the condition with which they are associated.

It must be borne in mind that when an individual who is the subject of primary angina pectoris suffers also from some other illness, either acute or chronic, both forms of angina may be present.

In these cases, the history, and a careful analysis of the associated symptoms will generally suffice to clear up the diagnosis.

Such terms as "pseudo-angina" or "mock angina" are misleading and improper.

Lack of appreciation of this distinction has led to the employment of such terms as "pseudo-angina pectoris" and "mock angina." It has followed that, when cases have been diagnosed as belonging to these groups, little consideration has been given to them. Such terms should not be used, for there is no such thing in nature as a false or an imitation disease. The fact that there is a superficial resemblance between two conditions gives us no right to call one a real disease and the other a false disease. That attitude is illogical. The symptoms provoked in either case have a real foundation,

and it is our duty to search for the real causes and not to cloak our ignorance by a vain and empiric nomenclature.

Prognosis and treatment in secondary angina.

The prognosis of cases with a hypersensitive nervous system is generally very good. The great majority of these cases are women between thirty-five and fifty years of age, and, after fifty, the tendency is for the painful attacks to abate. In many instances a surprising degree of strength is regained, and these patients may reach a very old age. It is scarcely necessary to point out that the removal of provoking causes renders the outlook brighter.

In the treatment of these hypersensitive cases, provoking causes should therefore always be searched for, and, as far as possible, removed. A change of environment, a peaceful, restful holiday—not a rest cure—is often of the greatest benefit. In the cases where it is impossible to remove the cause mild sedatives are of more avail than “cardiac tonics.” Bromides, especially the bromide of ammonium, pushed until a slight degree of apathy with sleepiness is produced, are often of the greatest benefit.

Treatment of primary angina.

The conception of angina pectoris as an expression of exhausted heart muscle affords principles on which to construct a rational treatment.

If relief from pain is to be attained, the heart must be relieved of at least a portion of its work, by judicious restriction of effort, or by complete rest, according to circumstances. Individual cases vary within wide limits, in regard to the amount of physical effort which can be undertaken without distress. Each case must be assessed on its own merits, and the amount of exercise regulated in accordance with the patient's capacity.

The amount of effort necessary to produce pain must

be defined. Some, it will be found, can go only a very short distance without inducing pain; in others pain only supervenes when a considerable effort has been undertaken. Again, some cases can walk a considerable distance if an easy pace be maintained, but develop pain immediately if the pace be increased.

Others find themselves pulled up by pain after quite a slight effort. On resuming the effort, *e. g.* walking, they are pulled up again. After two or three short efforts and rests they are able to go on, sometimes for a considerable time without further disturbance.

The onset of pain is influenced by various factors which require to be taken into account.

There are many circumstances which predispose to the occurrence of pain in susceptible individuals. Among these the effect of cold is important.

Most people who suffer from angina pectoris find their capacity for effort distinctly less in cold weather.

On going out on a frosty morning they are pulled up much sooner than usual.

The effect of food on the facility with which pain is induced is often very striking. So much is this so that the complaint for which these patients seek advice is often that of "indigestion."

In all cases in which pain after food is complained of by elderly people, especially elderly men, a careful enquiry should be made into the site and radiation of the pain, and the response to effort.

In one case under observation at the St. Andrew's Institute the patient, a man sixty-one years of age, finds himself pulled up by pain after walking fifty yards after a light breakfast. Before breakfast he can walk or cycle at an easy pace from one to three miles.

Other contributory causes, such as sleeplessness, gastric disturbances, tobacco, must be sought for, and, as far as possible, eliminated.

If the attacks tend to recur in spite of rest and the

removal of provocative causes, the more powerful sedatives, such as chloral and opium, must be employed.

In all cases during the attacks of pain, speedily acting vaso-dilators, such as whisky or brandy, alone or in hot water, and the nitrites (nitrite of amyl, nitro-glycerin) should be given.

In regard to the use of the nitrites a fear of over-dosage is sometimes expressed.

The effect of these drugs is so transient that there is no danger attending their use, and the dosage should be steadily increased till a full effect has been obtained.

CHAPTER V

BREATHLESSNESS AND ANGINA PECTORIS

It may be asked why most cases show heart exhaustion by breathlessness and not by pain. The production of pain in visceral disease requires a stimulus of a certain elusive kind, which we are yet far from interpreting. We know how extensive visceral disease may be, while pain or other sensory phenomena may be absent, as in ulceration of the lungs, stomach, and bowels. In other cases a small gastric ulcer may give rise to great suffering. So in the heart, seemingly, like diseases may give rise in one case to pain and in another to no pain.

But with diseases of the heart there is always evidence of functional inefficiency, and this is prominently brought out by breathlessness on exertion, or, in advanced failure, at rest.

It has, however, been a matter of observation that patients pulled up by pain on making exertion do not always suffer from breathlessness. In studying a great number of cases of angina pectoris, I find that a certain proportion give a history of a gradual limitation of the field of response to effort by the early onset of breathlessness. Subsequently those are seized with pain, and they are then pulled up by the pain and not by the breathlessness. On inquiry, I have always found that the pain came on after a less amount of effort than had pulled them up before by the breathlessness. I would suggest that in a measure the check to effort occurs according to which mechanism is first brought into action. This view is supported by watching certain cases of angina pectoris. In some, as the disease progresses, the ten-

dency to breathlessness increases, so that the patient can make but little effort without dyspnœa. In such cases, Cheyne-Stokes respiration is apt to occur, while the heart dilates and dropsy supervenes, and the attacks of angina completely disappear.

This also seems to account for the infrequency of angina pectoris with auricular fibrillation. When the latter condition is present, breathlessness is very easily provoked when the heart begins to fail. I have noted the onset of auricular fibrillation in cases of angina pectoris, and when this occurred they were much sooner pulled up by breathlessness on effort than they used to be by pain, and expressed great satisfaction at the freedom from pain, though distressed by breathlessness. Needless to say, their power of response always remained extremely limited.

Thus a man, aged sixty-six, consulted me for angina pectoris, which pulled him up on walking a short distance. After treatment he improved and was able to lead a fairly active life, though at times sharply pulled up by pain when he was walking fast. When seventy-three years of age the attacks of pain had become more easily provoked, till suddenly they ceased, and he found instead that he was very short of breath. Some months later he consulted me and I found he had auricular fibrillation. His statement was very characteristic. A few months earlier he was able to walk two or three hundred yards before being pulled up by pain. Latterly he could not walk a hundred yards without having to stop because of breathlessness. The heart was then rapid and irregular. He improved under digitalis, till he was seized with a cerebral embolism from which he died.

CHAPTER VI

SENSE OF EXHAUSTION

The sense of exhaustion is produced by a vaso-motor mechanism, and is not of cardiac origin, though it may be associated with cardiac inefficiency.

THERE are three kinds of sensations that most frequently indicate to the individual that his strength is exhausted—breathlessness, pain, and exhaustion. I have already described the first two and have indicated their cardiac origin.

I deal now with the third sensation—the sense of exhaustion. This is the most common of all sensations resulting from effort. Though often associated with grave conditions, it is frequently produced when there is no serious trouble. The sensation is one of limpness, the legs feel as if they would not carry the body, the brain feels fatigued, and the individual may become dizzy and stagger, and, in extreme cases, feel compelled to lie down to prevent an attack of faintness, and syncope may occur. This sensation varies greatly in degree, but it is so common that probably everyone has experienced it at some time or other—as, for instance, after first getting up after a more or less severe illness. Where, however, it generally presents itself is in people engaged with their affairs. It will then show itself in various guises, but most frequently as something amiss with the circulatory system. As this sensation may be associated with certain cardiac phenomena, such as breathlessness on exertion, palpitation, irregular action of the heart, murmurs, or increase in the size of the heart, the tendency is to refer the sensation to insufficient cardiac

power. It will, therefore, be worth while to consider the nature of this complaint.

To do this, one must find out the conditions under which it is provoked, and recovered from. To begin with, the sensation is usually not constant. If provoked by exertion, it will be found to vary from day to day, or there may be periods of weeks when the patient is free from it, and then weeks when it is easily provoked. Some days, if the patient walks a short distance, as on a hot and close day, too heavily clad, the sensation is readily provoked; on other days, when the sun is bright, the air cool, and the individual lightly clad, a wonderful amount of exercise may be taken without discomfort. Certain other states besides exertion may bring it on, as standing in a warm room. Many women feel faint and exhausted when having a dress fitted.

As the sensation is frequently present with cardiac abnormalities, it leads to the supposition that the condition is cardiac, but there are a few features which I regard as indicating that it is not directly cardiac in origin. The first is the inconstancy of the sensation. The effort that produces distress from a cardiac affection does so in almost every case. This follows from what I have said of the response to effort as the standard of cardiac efficiency. This variability in the production of exhaustion renders the possibility of a cardiac origin extremely unlikely. Another point to bear in mind is, that the cause that produces this exhaustion may also affect the heart, and produce, secondarily, an abnormal sign. To appreciate the origin of this complaint it is necessary to search for other signs. In many of those people who suffer from periods of exhaustion we find other abnormal signs referable to the vaso-motor system. Thus, many will show changes in the peripheral circulation, the hands and feet often becoming cold, not only in cold weather, but in response to some mental stimulus, such as the excitement of being examined. Many shudder at the idea of a cold bath,

as the after-reaction is one, not of a warm congenial glow, but of a shrivelled-up, cold feeling. In some the fingers become pale and cold, with diminished sensibility and a sensation of numbness. At other times the hands and nose become red. Some, in a warm atmosphere, become flushed and disagreeably hot. Not infrequently, pressure, applied to the abdomen, will cause the jugular veins to become full. This is an undoubted sign of the over-filling of the large abdominal veins, pressure on them filling the right heart, and impeding the blood-flow in the jugular veins. In healthy people one gets no such reaction, or only a very slight one, while in those in whom the exhaustion is easily provoked, the swelling of the jugular vein, on pressure of the abdomen, is sometimes very great. The blood-pressure in these cases is usually rather low, particularly during the attacks of exhaustion. It may rise as soon as the patient lies down or after the exhaustion passes off, and this accounts, probably, for the fact that sitting or lying speedily dissipates the sense of exhaustion.

Exhaustion is brought about by a depletion of the higher nervous centres.

Taking all these facts into consideration, I think we are justified in referring the state of exhaustion to a depletion of the higher nervous centres. The vaso-motor system is over-susceptible to stimulation, in the one case producing an over-action in response to cold, in the other case, to a widening of the peripheral field from warmth or exertion. In many cases the wearing of an abdominal belt, that firmly grasps the abdomen, will prevent the attacks of exhaustion and faintness. I have seen one man in whom this tendency to exhaustion and giddiness was so great that he could not follow his work and was only comfortable when lying down. A few minutes after standing up the heart raced off at a great rate and he became giddy and faint. A firm

abdominal belt at once produced such relief that, so long as he wore it, he was able to work. Another man, who consulted me, staggered and reeled and sometimes fell on standing up. He had made a habit of bending down and squeezing his belly before standing up. I found that the pressure in the abdomen caused great fullness of the jugular veins.

The provoking agent in the production of exhaustion is frequently a toxin.

It is not always easy to find the real cause of this condition, although there is now enough evidence to show that, in many cases, it is due to some toxic influence. A very considerable proportion of those who suffer so readily from exhaustion have distinct evidence of gastro-intestinal troubles. Many complain of different signs of dyspepsia, and we can often detect evidence of stasis in some portion of the intestinal tract, even when there is no complaint made by the patient of indigestion. Constipation is often present. Many years ago I was struck by the disappearance of those characteristic vasomotor phenomena (cold hands and feet) in a man with a chronic duodenal ulcer, on whom a gastro-enterostomy had been performed.

The heart is often irritable and causes a good deal of distress from attacks of palpitation, or from extrasystoles. In some it may dilate, and even show signs of insufficiency, in the sense that effort may readily produce distress or discomfort from breathlessness or palpitation. The mental condition may be affected; irritability of temper and depression are not uncommon. In many nutrition is impaired, with consequent loss of weight. The skin becomes dirty and yellow, especially in the armpits and on the abdomen.

Although the absorption of the products of decomposition from the intestines is probably the most common cause, absorption from other centres of infection is possible, as from the teeth and obscure inflammatory

conditions, like chronic appendicitis. A typical form is seen after exhausting illnesses, such as typhoid fever or influenza. In fact, any prolonged bacterial infection may give rise to it.

In all cases, therefore, when exhaustion is the chief complaint, causes other than the heart must be diligently sought for. (See Chapter XVI.)

CHAPTER VII

THE EXAMINATION OF THE PATIENT

The methods essential to the proper examination of a patient have hitherto not been utilised.

EVERY physician of experience works out for himself a method of examination, and though the methods of different physicians may vary, yet the object is the same in every case, namely, to get as much knowledge as possible of the patient's complaint. Apart from slight variations, there is a tendency for methods to become stereotyped, especially in the examination for the physical signs of disease. The methods of examination, as commonly taught and pursued, are often assumed to be so complete that no further progress can be made unless by the employment of some new mechanical means—so that it is asserted there can be no further advance in the examination of the patient by the unaided senses. The latter statement I have heard repeatedly made by physicians of the greatest eminence. As opposed to this view, I hope to demonstrate that knowledge essential to the understanding of the patient's complaint can be made out by the unaided senses, that artificial and mechanical aids can, in the nature of things, only be accessory aids, and that there is a great field for observation scarcely yet touched. Many are under the impression that modern education in the methods of examination is so exact and thorough that it cannot be improved upon. A friend of mine, a professor in one of our largest schools of medicine, told me with great pride how marvellously thorough was the teaching of the physical signs in his school. The students were taught to inspect, to palpate, to percuss, and to

auscultate with the greatest precision, and steps were taken by various means to prove their wonderful accuracy. This method of teaching, which has all the semblance of completeness, really fails because it does not go far enough. It stops short of taking account of certain essential factors, without which it is impossible to arrive at an adequate comprehension of the patient's condition.

Having considered the part that heart failure plays in any given case, and having indicated the nature of the symptoms by which its presence can be revealed, the importance of the patient's sensations in obtaining a knowledge of the heart's efficiency will be readily recognised. Hitherto, in books on diagnosis, nearly all the stress has been laid on the physical examination, and little time given to the elucidation of the patient's sensations.

To obtain the necessary information, one must proceed by question and answer. Questions must be framed so that the patient recognises what is asked for, and it is, therefore, incumbent upon the examiner to have a clear conception, in his own mind, of the kind of information he is seeking. Each question, therefore, must have a definite object, and must be put so clearly that the patient comprehends its meaning; it is necessary also to insist upon the answer being limited to the question, and as full an answer as the patient is capable of giving must be obtained before the question is left. Now, this implies, in the first place, a knowledge of the kind of information the patient is capable of yielding, and, in the second place, that much training is necessary before one can attain the skill of a competent examiner.

When the patient and physician come first together, it often happens that there is an unconscious struggle who is to be dominant. Many patients come full of their own ideas about their complaints, with theories as to the nature and cause of their sensations, and eager to impart their own opinions. Or they come

with a bundle of notes which they insist upon reading. The first question put to them: "Of what do you complain?" will immediately give them an opening, and they will, if not checked, proceed to indulge in an elaborate history of their troubles. This must be quietly and firmly repressed. The story of their life must be reserved until the examination is finished, and their replies must be limited to the sense of the question asked. If this is done, in a few minutes all the essential facts will have been obtained, and if then they are asked: "Is there any other point you have not mentioned?" they will gaze helplessly and, in a surprised tone, acknowledge that all the facts have been obtained in five minutes, which they thought would take half an hour to tell.

When the patient presents himself for examination, the physician naturally scrutinises him and makes a mental note of his gait, his facial aspect, and how he comports himself generally. The first question bearing upon his condition should naturally be to ask of what he complains. The reply to this question should be limited to the sensations which immediately caused him to seek advice. Thus, if it is a heart trouble, he must clearly specify the sensation he himself experiences, as pain, breathlessness, or exhaustion. A note should first be made of all the sensations of distress or discomfort of which he complains and each sensation taken up by itself. Thus, if pain is complained of, the situation in which the pain first appeared must be noted, getting the patient to indicate this by placing his hand over the region. The direction and extent of its radiation must next be defined. The character of the pain must then be inquired into, as to its intensity and duration—whether continuous or intermittent. Finally, the circumstances which provoke the pain must be recognised, whether it is brought on by effort, mental excitement, or when the body is at rest, and its relation to food. With these facts clearly appreciated,

the time of the first occurrence of the pain, even in its mildest form, should be determined, together with the occasion of its appearance. Exhaustive inquiries of this sort should be directed to every other sensation, naturally modifying the question according to the character of the sensations. With the facts thus elicited, a fair perception of the main facts relating to the complaint will have been obtained, and the inquiry then follows as to illnesses and habits of life that may have induced it. A brief inquiry into the functional efficiency of other organs—as sleep, the state of the digestion, menstrual functions, and frequency of micturition should also be made.

As distinct from these inquiries, an inquiry must be made in all cases of heart trouble as to the efficiency of the circulatory system, by asking the patient the amount of effort he is capable of undertaking without distress, how far he can walk, whether his work entails bodily effort and, if so, how it affects him; what games he plays and how he bears the effort. Where there are evidences of limitation, the nature of the sensations which induce the limitation must be elicited—as breathlessness, palpitation, pain, sense of exhaustion. From the character of these sensations one can gather whether the story of the patient is one of heart failure or of vaso-motor disturbance.

In children some difficulty may be experienced in eliciting the facts, but, as a rule, it is easy to appreciate the heart's efficiency when they are old enough to talk and run about. Unfortunately, there is a mistaken idea in the minds of parents and physicians that a suspicion of heart trouble demands that a child should be kept at rest. No doubt, in rare instances of acute or progressive lesion, rest is necessary, and the need for it may be recognised by a physical examination. But a great number of children, who are supposed to have weak or impaired hearts and are able to run about, are often needlessly restricted, because of some

physical sign—murmur or irregularity. While I shall deal with the phenomena in detail, it may be noted here that a child with an impaired heart is not likely to injure it by over-exertion. Children are very sensitive to distress provoked by cardiac inefficiency and will themselves abstain without being told, and one can usually recognise the degree of efficiency by asking the child if he likes running about with a hoop or running upstairs. When they say they like doing it and it gives rise to no distress, it may be safely assumed that, whatever the cause of the trouble, it has not impaired the heart's strength. I cannot recall a single instance where I could reasonably attribute heart failure to a child's voluntary exertions.

The same reasoning applies to the games of boys and girls; when they exert themselves and find pleasure in doing so, it may be taken for granted that no harm is being done.

CHAPTER VIII

THE HEART FROM YOUTH TO OLD AGE

SUFFICIENT attention has not been directed to ascertaining what the manifestations of the heart may be in health. Before dealing with the physical signs, it is necessary to say a few words about the variations that are to be found in healthy hearts at different periods of life. Perhaps there is no fallacy so fondly cherished by the profession as that a heart, to be normal and healthy, must be of a certain size, have the rhythm of its beat regular, and its sounds clearly struck and free from any suspicion of modification. The presence of a murmur is often considered to be inconsistent with the idea of a healthy heart. True, there are some who are willing to admit that this view is not justified in every case; but the great bulk of the profession, and the teachers of the profession, do adhere to this view, if not in theory, at least in practice. In healthy young folks, from the time of birth, the rhythm of the heart may show physiological variations. The young, healthy heart is extremely sensitive to stimulation; on account of its peculiar, sensitive mechanism, we may have marked variations in size, in rate, in rhythm, and in the sounds of the heart. It behoves us, in any given case, when we find a departure from that standard which has hitherto, and foolishly, been taken as the standard of a healthy heart, to inquire if the unusual sign is not, after all, a purely physiological manifestation in a healthy heart. Having asked that question, one is naturally spurred on to seek for those signs which will decide it.

This does not apply only to young subjects. In later

life, too, there are many departures from the standard which are in perfect accord with the idea of a healthy organ. These departures are different from those met with in early life, and therefore, here again, the first question to be asked is, "Is this sign a manifestation of changes which do not indicate a disease, nor embarrass the heart in its work, but which are, in fact, the changes that accompany advancing years?" As years go on, changes occur in the outer man which are visible to the naked eye, and we know that changes of a similar nature may take place in the organs hidden from view. A slight thickening of the edges of the fine texture of the valves may modify the sounds of the heart without interfering with its efficiency, so that murmurs are quite a common occurrence in people of middle age who lead strenuous lives, yet who never suffer from any form of heart failure.

Changes of a slight nature may lead to disturbance in the rhythm of the heart, and even to alterations in its size; yet the heart may not be impaired. There are thus numerous changes, of a kind readily perceptible, which are of no more significance than grey hairs. It behoves us then, in valuing an unknown phenomenon, to pause and ask whether or not it is but a physiological accompaniment of advancing years. If it is a physiological accompaniment it cannot be taken as an indication of disease. This truth will be kept in mind in describing the individual phenomena; I mention it here to insist upon the necessity of asking such questions when confronted with an abnormal sign.

CHAPTER IX

MURMURS : PHYSIOLOGICAL, FUNCTIONAL, AND ORGANIC

Murmurs were assumed to be of serious significance before their real nature was understood.

ABOUT a hundred years ago auscultation began to be systematically employed in the examination of the heart. With that injudicious enthusiasm, which at all times has heralded a new method of observation, fabulous qualities were at first attributed to the stethoscope. People were found to have murmurs before their death; hence, at a time before the cause of the murmurs was known, and long before any single observer had watched individual cases long enough to understand the significance of the murmurs, the statement went forth with all the weight of the highest authorities that these signs betokened grievous heart trouble. To-day, notwithstanding the enormous amount of attention which has been given to the subject, the whole profession suffers from this untrustworthy observation. Perfectly healthy men are, to-day, being rejected for the Army, or invalided out of it, because a murmur has been detected in their hearts. Others, who present themselves for life insurance, are rejected or made to pay a higher premium for the same reason, while innumerable individuals are subjected to prolonged treatment and great restrictions in their mode of life because these early superficial observations have misled the profession. At times, physicians of experience will admit that certain murmurs may not have a serious significance; in saying this many of them appear to imagine that they have made an important contribution to the subject. But having failed to point out at the same time how a murmur can be valued so as to discover when it is of significance and when it is not, they have

really failed to carry forward our knowledge in any respect. The result of this imperfect comprehension of its meaning is that, at present, every graduate leaves hospital with but the vaguest notion how to assess the value of a murmur. In other words, teachers and writers have hitherto shrunk from laying down clear and definite lines for guidance, and I know that, in attempting to do this, I am undertaking a very hazardous enterprise. I do so, however, not with the idea that I have finally settled the matter, but in the hope that others, who follow me, may test my observations, and so extend the knowledge of this very important subject.

It must be many years since Graham Steel startled me out of a deeply-cherished belief by stating that "no one ever dies from mitral regurgitation." By this he meant that, when the heart failed and there was a mitral systolic murmur present, heart failure had occurred, not because of the regurgitation, but because there were present other factors which provoked it, such as some myocardial disease or impairment. This was so opposed to the conception of heart failure by back pressure which I had been taught to accept that I carefully observed my patients to see whether or not it was true; and now I can fully endorse Graham Steel's dictum. I have kept records of great numbers of people with murmurs and watched them for years; I have observed carefully and have noted that some men have pursued laborious lives and have shown no sign of heart failure, while others have fallen ill and died. From this long research I have drawn certain conclusions to which I shall now refer.

Murmurs may be physiological as well as functional and organic in origin.

I use the term physiological in the following sense : Murmurs may be present in hearts which are perfectly healthy in every respect. The conclusion that these

hearts are healthy is arrived at when the efficiency of the heart as estimated by its capacity to respond to effort is unimpaired. The individuals in question may lead strenuous lives and never show any sign of heart failure, while, if by accident or other cause they die, no disease or damage can be detected in the heart on post-mortem examination. If a large number of the healthy young of both sexes, during the second and third decade of life, be examined, a large percentage will be found to show murmurs. The assumption, then, that the sounds of a healthy heart must be clearly struck and free from murmurs is not based on accurate observation.

The term "functional murmurs" has passed into common use, and is used to include murmurs which are not due to organic defects of the valves. The term is often made to include physiological murmurs, and as there is implied in it the idea of some derangement, it is very apt to be assumed that the "functional" murmur indicates impairment. In this way the physiological murmur comes to be regarded as a pathological condition. Thus, in dilatation of the heart, a murmur may be present; it is assumed that the murmur is caused by a modification of the muscle apparatus that regulates the size of the orifice, and that, as a result of this modification, a leakage occurs. There is every reason to suppose that this view of the cause of certain murmurs is justified, and it may not always be possible to distinguish between the physiological and functional murmurs. Consideration of the functional efficiency of the heart offers, undoubtedly, the best guide in these cases, and we may fairly assume that when the heart is normal in size and its efficiency is not impaired the murmur is physiological. When, on the contrary, there is an increase in the size of the heart, with limitation of the heart's response to effort, we may assume that the murmur is functional or organic.

During a febrile illness functional murmurs frequently appear and they are never of much moment. It is

difficult, however, to distinguish them from murmurs due to an actual lesion of the valves unless certain characteristics peculiar to organic murmurs are present.

Functional murmurs may persist after the subsidence of the fever, or physiological murmurs may then appear; and there may consequently be some doubt whether, after all, the murmurs may not be due to a lesion of the valves. The absence of other signs of cardiac disturbance, such as increased rate and size, may in these cases be taken as an assurance that the murmur is not organic, while the appearance of the youthful type of irregularity, especially if it is well marked, will set the matter at rest by indicating that the heart has escaped damage. I deal with this question of youthful irregularity very fully in Chapter XII.

How physiological and functional murmurs may be recognised.

These murmurs always occur during the systole of the ventricle. In a large number of people they are unaccompanied by any other sign of cardiac abnormality. A great many people, especially young people, who have this systolic murmur never afterwards show any sign of cardiac inefficiency, though no treatment or care has been undertaken. Post-mortem examination of such hearts shows absolutely healthy valves. In some the murmur is present when standing and disappears when lying down. In others the contrary happens, the murmur being present when lying down and disappearing on standing. In some cases it may not be present when the heart increases in rate during exertion or during inspiration, but may appear as the rate decreases after exertion and excitement or during expiration. In many it is persistent under all circumstances. It would be unprofitable, in our present state of knowledge, to inquire into the reason for these variations. We are so ignorant of the factors concerned in the production of such murmurs that our conclusions would be but guess-work.

What is essential to us as physicians is to recognise that a systolic murmur, whether variable or persistent, may be of no significance as far as the future of the patient is concerned, and that when it is the only abnormal sign present, and the response to effort is good, it implies neither cardiac disease nor cardiac impairment.

If there be evidence of the dilatation of the heart, then probably the murmur is functional, but, as I have said, this point is of little significance; our opinion has to be based on the degree of heart failure and the circumstances inducing the dilatation. In anæmia, and conditions inducing exhaustion, functional systolic murmurs are frequent, and here also it is manifest that the murmur is of little significance.

How to estimate the value of systolic murmurs of organic origin.

It is not always possible to tell whether or not a systolic murmur is due to an actual disease of the valves. But when we are in doubt we can estimate its value by consideration of the heart's efficiency and the presence of other signs; for it must be borne in mind that mitral regurgitation, even from a damaged valve, is seldom, if ever, of much importance. It is not the regurgitation which is the serious matter, but the fact that a damaged valve indicates that the heart has been invaded by disease. The question arises, Has the disease affected more vital parts? Rough character in a murmur, and especially a musical quality, may be taken as an indication of a damaged valve. But it must be remembered at the same time that organic murmurs may be of a soft, blowing character, and thus be indistinguishable from those of functional origin. In order to estimate its value the organic murmur should be considered from the following points of view: (1) Condition of the orifice. The presence of the murmur may be associated with signs indicating such narrowing of an

orifice (as, for example, the aortic) that it is clear that the ventricle is being embarrassed in its work. If this has happened the response to effort will be limited and the ventricle will be enlarged. It is on these two factors that we must base our estimate of the condition of the heart. As I have said, it is doubtful whether mitral incompetence *itself* ever constitutes a serious embarrassment to the heart. (2) Condition of the myocardium. The presence of this systolic murmur may indicate that the heart has been invaded by a disease, and, as the disease is rarely limited to one structure, it will be necessary, in all cases, to consider whether any damage has been done to such other structures as the myocardium and the pericardium. In the absence of any increase in the size of the heart, and of any abnormal action, and in the presence of a good response to effort, it may be assumed that the damage is so slight that it does not embarrass the heart in its work. On the other hand, if impairment of the heart's efficiency be present, and there are such other signs as increased size and abnormal rate and rhythm, our opinion must be based on those other signs as well as on the murmurs. We must, in short, see the condition as a whole.

The reasons for estimating the value of murmurs in this manner.

The reason underlying this attitude becomes very plain as soon as it is understood in what way heart failure is produced. If a murmur is caused by a lesion which embarrasses a chamber in its work, then that chamber will alter in form either by dilating or by becoming hypertrophied. The absence, then, of any alteration in the size of the heart is evidence that there is no embarrassment. Moreover, a lesion embarrassing the heart in its work would also render the organ less efficient, so that an inquiry into the cardiac efficiency and a knowledge of its exact state furnish us with important material upon which to base our opinion.

The meaning of systolic murmurs when these are accompaniments of heart failure.

When a systolic murmur is present along with heart failure we invariably find other evidences of cardiac trouble. These other evidences are important guides; they comprise an increased size of the heart, the presence of an abnormal rhythm (for example, auricular fibrillation), and changes in the manner in which the circulation is maintained.

We may meet with signs of extreme heart failure in which there is no increase in the size of the heart, but in which a systolic murmur is present. As an instance let me cite attacks of angina pectoris readily provoked. It may be taken for granted that in these cases the murmur is incidental and that its cause has little or no part in the production of heart failure, though its presence may indicate that the same changes which may have affected the valve are present in other structures the impairment of which has induced the heart failure. It is not always possible to state definitely what is the exact cause of a given systolic murmur. This should be remembered.

In speaking of systolic murmurs I have not sought to differentiate between the various orifices at which they may arise. The mitral systolic murmur—the murmur heard loudest at the apex and propagated towards the axilla—is well recognised. Systolic murmurs heard at the base cannot be identified with certainty, and much dispute continues about the origin of many of these murmurs. When, in the absence of the diastolic aortic murmur, there is a possibility that the systolic murmur may be aortic in origin, the important question is whether or not there are present signs pointing to a marked narrowing of the aortic orifice. If these signs are absent, little attention need be paid to the systolic murmur. If narrowing of the aortic orifice be present, the left ventricle will be embarrassed, a state which will be revealed by increase in its size accompanied by a diminution of

the heart's power of response to effort. Other signs may be present also, as a thrill over the region of the aortic valves, propagation of the murmur into the carotid, and a small anacrotic radial pulse of low tension.

Extreme aortic stenosis without regurgitation is a rare condition, and in the few people I have seen who showed it, it has occurred towards the end of life. In these cases the heart has been grievously exhausted—breathlessness or pain being easily induced. There is usually a syphilitic history, except in the senile cases.

The meaning of a systolic murmur in acute febrile conditions.

It is in acute or febrile states that the greatest difficulties of diagnosis arise. The elevation of temperature or the toxins produced by a microbic invasion of some other part of the body may readily alter the muscular mechanism of the heart in various ways and so produce a functional systolic murmur. On the other hand, the microbic invasion may attack the heart itself and so damage the valve directly and produce a systolic murmur. It may be difficult to determine which of these two conditions is present, and it is often necessary to wait till the fever has subsided in order to arrive at certainty. If the murmur changes its character from day to day, tending to become more marked, and, especially, if it takes on a musical quality, we may be fairly sure that the disease has affected the valve. Again, if, after the subsidence of the fever, the heart remains persistently increased in rate, suspicion that the valve is damaged is strengthened. The occurrence of such other signs, as slight heart-block, will further testify that the heart itself is the seat of invasion. On the other hand, return of the rate to the normal after the subsidence of the fever, especially when this is accompanied by the appearance of the youthful type of irregularity, will indicate, and this in spite of the systolic murmur, that the heart has escaped infection. It is scarcely necessary to insist on the importance of this view in regard to such diseases as rheumatic fever.

CHAPTER X

MURMURS (*continued*)

DIASTOLIC MURMURS

(Mitral Stenosis and Aortic Regurgitation)

The murmurs which indicate lesions of valves embarrassing the heart and leading to heart failure occur mainly during the diastole of the left ventricle, and of these there are two which include the great majority of serious valvular affections, namely the murmurs due to stenosis of the mitral valve, and the murmurs of aortic regurgitation.

Mitral Stenosis.

When the murmurs of mitral stenosis appear, and how they alter.

THE most instructive murmurs are those produced by stenosis of the mitral valve. Yet symptoms evoked by this valvular affection, and its accompanying changes in the heart muscle, are far from being properly appreciated and understood at the present day. In the study of these cases we must disregard much of the traditional teaching and approach the subject with an open mind, recognising especially the fact that here there is something far beyond the mere presence of a particular murmur.

In a number of people whom I have attended for rheumatic fever I have been able, during some ten, fifteen, or twenty years, to observe and to note the changes that gradually arise in the heart as a result of this affection. In none of these cases was there ever any indication, during the causative illness or convalescence, that mitral stenosis was present. Some recovered with a seemingly intact heart, others were

left with a mitral systolic murmur. It was not until years had elapsed that the first faint signs of stenosis became apparent. In one woman I perceived, as the earliest indication of the condition, a short thrill preceding the first sound. Some months later I detected a short presystolic murmur which was not always present and which usually appeared only when the heart was excited to violent action. As time went on this murmur became constant and gradually increased in length, starting in late diastole of the ventricle and running up to the first sound—the characteristic crescendo of the presystolic murmur of mitral stenosis. Associated with this state of affairs there was a doubling of the second sound, but I am not certain as to the exact time of the first appearance of that phenomenon. This is the stage at which we see most cases of what is called early mitral stenosis, for the patient has hitherto been leading the life of a healthy person with a healthy heart, and only becomes alarmed when subjective signs of heart failure begin to show themselves. The next change is the appearance of a murmur immediately after the second sound, at the beginning of ventricular diastole. As time goes on this murmur lengthens, and we note that it begins with a high pitch and decreases in loudness—the diminuendo murmur of mitral stenosis. The diastolic murmur may lengthen and run up to the presystolic murmur, with the result that the whole diastolic interval is filled by it. It begins loudly after the second sound, falls slightly, and then increases in loudness and ends in the first sound. During all this time the heart is usually perfectly regular in rhythm (Fig. 7). This stage may last for many years.

The sudden onset of persistent irregularity is accompanied by disappearance of all signs of auricular contraction, the auricle having passed into fibrillation.

In the majority of people the next change takes place with dramatic suddenness, the presystolic element of

the murmur disappears suddenly and entirely, and only the diastolic portion remains. At the same time the heart becomes very irregular in its rhythm and frequently there is severe heart failure (Fig. 8). Now it is this stage which is so instructive and which is of such great importance in relation to a due appreciation of the cause

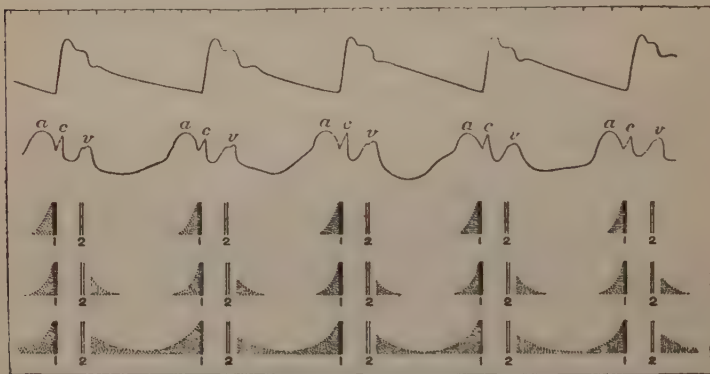


FIG. 7.

Tracings of the radial and jugular pulse with diagrammatic representation of the changes in the murmurs of mitral stenosis. The down strokes 1 and 2 represent the first and second sounds of the heart. The first indication of stenosis is the appearance of the murmur before the first sound, and on reference to the jugular tracing this murmur will be found to coincide in time with the wave, *a*, due to the systole of the auricle. With an increase in the stenosis, a short diastolic murmur appears, immediately after the second sound, and this is represented in the middle diagram. A further increase in the stenosis, shown in the lowest diagram, leads to prolongation of the diastolic murmur, which in combination with the presystolic murmur fills up the whole interval between the second and first sounds. (For the next change see Fig. 8.)

of heart failure in many instances. It was in 1897 that I was able to establish the relationships of a series of phenomena which may be observed at this stage of the condition. I attended a woman for rheumatic fever in 1880, and kept a watch over her till her death in 1898. About ten years before her death she showed signs of mitral and tricuspid stenosis, her heart being regular

in rhythm, with the exception of ventricular extrasystoles on rare occasions. Suddenly she began to suffer from extreme heart failure, and I found that her heart was irregular and that the presystolic mitral and tricuspid murmurs had disappeared. The auricular waves

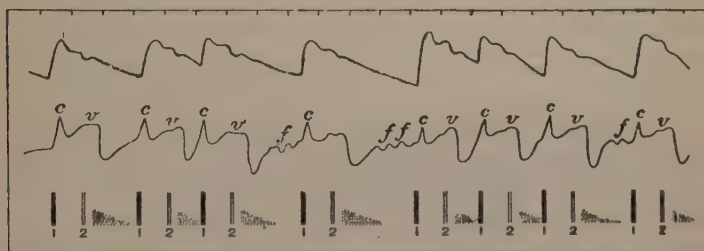


FIG. 8.

After the changes described in Fig. 7 the next change is a sudden alteration of the rhythm of the heart. In Fig. 7 the heart is quite regular; in Fig. 8 it is irregular. The wave, *a*, which is a marked feature in the jugular tracing of Fig. 7, is absent in Fig. 8. The presystolic portion of the murmur is also absent in Fig. 8, this feature being best appreciated during the longer pauses of the irregularity. The disappearance of these evidences of auricular activity and the irregular action of the heart indicate the onset of auricular fibrillation. The waves *ff* are due to the fibrillating auricle.

which had been present in jugular and liver tracings had also disappeared. In fact, all evidence of auricular systole had suddenly ceased. The irregular action of her heart persisted till the time of her death, and there was never a return of the presystolic murmur. (Compare Figs. 7 and 8.)

How an appreciation of the nature of the changes in the murmurs of mitral stenosis helps towards an accurate diagnosis.

To deal faithfully with these patients, it is necessary that the meaning of these changes be clearly comprehended. Mitral stenosis is almost invariably a progressive lesion and the impairment of the efficiency of the heart is also progressive. The rate of stenosis varies

—sometimes it is relatively slow, sometimes relatively fast—so that it is important to gain information regarding the rate of this progress as well as regarding the extent of the stenosis which may be present. This, therefore, is a point which must be dealt with in every case that presents itself. It is true we are unable to settle these questions with absolute precision; nevertheless we can arrive at an approximate estimate, which will help us greatly in giving advice.

In the case quoted above, I pointed out that a considerable period of time elapsed between the invasion of the heart by the rheumatic disease and the first appearance of the signs of stenosis. The explanation of this is that the damaged mitral valve was not at first stenosed, but was simply inflamed with, it may be, some destruction of its fine margins. Inflammation leads, in the process of healing, to the formation of fibrous tissue. As a usual consequence, the fibrous tissue, after a time, tends to contract. Hence the valvular orifice becomes increasingly narrowed. At first the narrowing gives rise to a short murmur at the time of auricular systole, which is evanescent, and appears only when the auricle is stimulated to extra effort. With an increase in the narrowing the murmur becomes permanent and prolonged. There are two periods during ventricular diastole at which the blood is sent into the ventricle with some degree of force. One of these, as we can easily understand, is the time at which auricular contraction takes place. The other period is not so widely recognised. During ventricular systole the blood returning from the lungs is stored up in the left auricle, and it fills the auricle and raises the pressure in that chamber. At the end of ventricular systole the sudden relaxation of the ventricular muscle causes a great fall in ventricular pressure—a fall to a pressure lower than that in the auricle; consequently, at the end of systole there is a rush of blood from the auricle to the ventricle. In hearts free from mitral stenosis the blood passes quite

silently, but, as a result of the narrowing of the orifice, the stream gradually becomes audible, at first giving rise to a slight murmur, later to a murmur which increases in volume and strength with the increase in narrowing. The inrush of blood is, of course, greatest immediately after ventricular systole and diminishes in quantity as the auricle empties, so that the murmur also diminishes in volume at this period and we get the *diminuendo* murmur.

Recognition of these facts enables us to understand that, when we find the diastolic murmur, the narrowing of the orifice has become so extreme as to afford for the passage of the blood little more than the characteristic slit with which all are familiar in the post-mortem appearance of advanced mitral stenosis.

When we inquire into the history of the case we can usually get an account of the attack of rheumatic fever which set up the valve mischief; the distance, in time, from that attack and the nature of the murmur itself afford data for determining the degree of stenosis present and the probable rate of progress.

This, I know, will be a very rough estimate; in many cases other factors will have to be taken into consideration, and some of these I will mention later. I tried myself to get more precise information, but I had not realised the value and possibilities of this method when my opportunities for more careful observation passed away. This, therefore, I would suggest, is one of the subjects that young practitioners might take up, for I am certain that in the course of work extending over fifteen or twenty years they should be able to add a most valuable chapter to our knowledge and thus to confer benefit upon our patients.

Even this limited application of the method, however, will be helpful in many cases. Mitral stenosis in the very young, accompanied by presystolic and diastolic murmurs, is a very grave condition indeed; whereas in a patient aged about forty, with a history of rheumatic

fever in childhood, and only a presystolic murmur present, the rate of stenosis is presumably slow and may be stationary. In one patient who came under my care with a very slight mitral stenosis at the age of fifty, having had rheumatic fever at eighteen, the murmur never altered in character, though I watched him for twenty-five years. He died at the age of eighty-two.

In most cases of mitral stenosis damage is done to the muscle wall. This may produce heart failure, auricular fibrillation and heart-block.

In these cases there are other factors present in addition to the mitral stenosis which modify the heart's action. In many cases the rheumatic infection is not limited to the valves, but has invaded other tissues. The muscle substance is not infrequently damaged, and when this is the case heart failure is readily induced because of changes going on in, and impairing the functional activity of, the heart muscle. Many of the young who suffer severely from heart failure display this condition, and in them the condition may lead to death, though it is wonderful how good the recovery from extreme heart failure, with dropsy and extreme dyspnœa, sometimes is in the young. I have repeatedly observed in these patients periods of heart failure, during which recovery seemed hopeless; yet, after a long period, improvement has set in, and the individual recovered so far as to be able to pursue even a laborious occupation. One can but conclude that some product of the rheumatic invasion has caused a temporary weakening of the heart muscle. Many serious cases of mitral stenosis in the young, however, never show heart failure with dropsy. In these cases breathlessness and palpitation are the most troublesome symptoms, and hæmorrhage from the lungs is not infrequent. As a rule, this state is a dangerous one. Yet, although increased pressure in the pul-

monary blood-vessels may be one mischievous factor, I must confess to being unable to explain the manner of the heart failure in these cases.

There is a stage in mitral stenosis which has long been recognised, though the reasons for its occurrence and its significance have not been understood till recent years. This stage is that in which the "mitral pulse" appears—a pulse frequently small and soft, with a very irregular rhythm. This pulse we now recognise as being due to auricular fibrillation.

I will defer the consideration of this subject till I deal with auricular fibrillation as a whole. The inception of this new rhythm alters the whole aspect of the case, not only in regard to the physiological change in the heart's action, but in regard to the heart's power of maintaining the circulation and in the peculiar response of the heart to drugs of the digitalis group. It appears also in many other conditions. In the meantime, I would merely remark that there exists good ground for assuming that the appearance of auricular fibrillation is not due directly to the valve lesion, but to a loss of the control normally exerted by the sino-auricular node upon the contraction of the auricle.

Auricular fibrillation is not the only evidence we have of damage other than valvular. There are, in addition, a number of peculiar phenomena related to the bundle of tissue which joins the auricles and the ventricles. The phenomena are concerned with the changes giving rise to heart-block. In heart-block we find the explanation of the mid-diastolic murmur. This murmur occurs in mitral stenosis, and is due to the systole of the auricle. In heart-block there is a lengthening of the interval between the systole of the auricle and that of the ventricle, due to the slower passage of the stimulus from auricle to ventricle along the damaged bundle. Sometimes we get ventricular beats dropping out when the stimulus fails—as it does occasionally—to pass along the bundle. This is not infrequent after the administration

of digitalis, for digitalis acts very readily on a damaged bundle (see pages 22 and 189).

In estimating the value of these peculiar phenomena we must not attach too much weight to any single one of them. With partial heart-block or with auricular fibrillation the outlook may still be fairly good, and individuals showing these signs may lead useful lives for many years.

In estimating the value, then, of any phenomenon, however peculiar, we must be guided mainly by the functional efficiency of the organ as tested by its response to effort.

Aortic Regurgitation.

Aortic regurgitation has a varied significance, and the manner of estimating its significance is discussed.

Seeing the bad cases of aortic diseases in the hospitals, I formed the opinion, which many of us must have formed, that aortic regurgitation is a very dangerous complaint. The writers of our standard text-books treat the subject with great gravity. This view was confirmed by my experience in a few cases shortly after I entered general practice. It was, therefore, with some surprise that, in those early days, I accidentally came across a carter, who had a broken leg and who had been leading a strenuous life, in whom I detected double aortic murmurs. The man declared he had never suffered from any inconvenience. I had occasion, some twenty-five years ago, to examine a colleague for life insurance, and I found that he, too, had double aortic murmurs. Without hesitation I described him as unfit for insurance. Since that time he has led a busy life in his practice and played strenuous games of tennis and cricket; the last I heard of him was a few years ago, when he called and informed me that he had obtained a commission in the Army. Though then over fifty years of age, he had never suffered from

the slightest symptoms of heart failure. In another patient, a woman of fifty-two who had had rheumatic fever at twenty, and who came under my care in 1892, there was present a well-marked diastolic aortic murmur and a typical collapsing pulse. She had lived an active life, and she continued to do so, though in her later years she had suffered at times from attacks of angina pectoris. In 1906 she developed auricular fibrillation, and died in 1907 at the age of sixty-seven.

Being brought into contact with many cases such as these, as well as other cases which speedily died, I asked myself the question: Why are there these great differences in the outlook in cases of aortic regurgitation, and how can I find evidence that will guide me to a rational prognosis in any given case?

I admit at once that I have not been able to answer this question satisfactorily, though I have obtained several facts that have helped me in many cases. I naturally thought at the beginning that the degree of danger would depend on the extent of regurgitation, and so I tried, by the study of the distribution of the sounds and murmurs of the heart, to find a basis for estimating the amount of regurgitation; but I failed to find any reliable facts. As far as can be judged from post-mortem examination, the extent of the damage to the valve does not necessarily correspond to the degree of heart failure. Valves which have proved to be only slightly damaged have, in my experience, been associated during life with severe heart failure.

I found a better guide in the size of the heart. In the cases I have mentioned, and in numerous others who led vigorous lives free from any suggestion of heart failure, the left ventricle was only slightly hypertrophied, the apex-beat being slight and located only just outside the nipple line. So that an important element for the prognosis is the degree of enlargement of the left ventricle.

But even when the ventricle is considerably hyper-

trophied, and a large, forcible apex-beat is found situated well outside the nipple-line, a life of moderate utility is often led by the patient, and this in spite of the fact that the response to effort is impaired. In these cases I found the rate an indication of some value, and was able to conclude that those whose hearts were distinctly enfeebled had a persistent increase in rate—about 90 when at rest, while the more vigorous had a rate approximating to the normal or under 80. Nevertheless, some discretion is needed, for many sufferers from aortic disease have extremely excitable hearts, the rate of which increases greatly when they come into the doctor's presence. They can tell this themselves, for the beat is very forcible and they are conscious of it. In these cases, when this violent action of the heart is detected, the patient's experience should be taken as the guide.

But the most important factor in arriving at a prognosis is the patient's response to effort. It may be taken for granted that, if the patient is unconscious of any discomfort or palpitation on severe effort, the outlook for him is good.

The state of the pulse affords some help. When an extremely collapsing pulse is felt, and when, on using the blood-pressure instrument, the systolic pressure is found to be very high, and the diastolic pressure very low (190–60), then the condition may be grave, and will be found to be associated with evidence of heart failure. A moderate difference between the systolic and diastolic pressure (180–90) is quite consistent with an efficient heart; while in some of these cases the difference is equal to the normal (140–85). Needless to say, in these last instances the pulse has little of a collapsing character. The high systolic pressure is, presumably, the cause of bleeding from the nose, which is not infrequent in aortic regurgitation.

In aortic regurgitation we often find that the vasomotor system is in a state of great sensitiveness, and

sudden rises of arterial pressure may be associated with severe attacks of angina pectoris. To this sensitive vaso-motor system may be attributed the attacks of syncope which are neither infrequent nor serious.

So far I have spoken of aortic regurgitation, which has been established for some time, and in which the disease of the valves is, presumably, stationary. These cases are usually rheumatic in origin, or in the young arise from some obscure infection. The aortic disease occurring in the middle-aged and elderly, which is due to atheromatous changes, seldom leads to serious heart failure.

Aortic disease due to syphilis is often progressive and of grave significance.

In syphilitic disease the aortic condition is often a very serious one. Once it has started, it often rapidly destroys the valves, and in the aorta itself it may seriously interfere with the blood supply of the heart by blocking the coronary arteries. If taken in time and treated energetically by anti-syphilitic drugs, the disease may be stopped, and a fair recovery may follow. In some cases, however, the disease extends rapidly, and death ensues within a year or two of its commencement.

When heart failure sets in with aortic regurgitation it is difficult to check its progress.

The heart failure of aortic regurgitation is in some respects peculiar. It is rare to find dropsy; indeed, when that is present the disease may be regarded as a complicated one, embracing, usually, among its manifestations an affection of the muscle wall.

Attacks of pain, often of the greatest severity, are a very frequent evidence of myocardial exhaustion, as also is violent beating of the heart; attacks of extreme dyspnœa are not infrequent, and death sometimes occurs during the first attack.

It is very doubtful if the enormous mass of muscle

which forms the ventricular walls in these cases, represents a genuine hypertrophy. It is more probable that some disease condition has taken part in the increase. The large muscle is very ineffective in maintaining the circulation, even when the leakage through the aortic valve has not seemed to be great.

When heart failure sets in to any considerable degree the condition is less susceptible to treatment, and recovery is less frequent and less pronounced than in heart failure resulting from any other condition. One frequently sees extraordinary recoveries from extreme heart failure—as, for example, in mitral stenosis—but one does not meet with such recoveries in aortic disease. Even when heart failure is extreme in auricular fibrillation with mitral stenosis, a good recovery may often occur, but in auricular fibrillation with aortic disease, when heart failure sets in, the patient steadily drifts, and we are seldom able to check the downward progress.

CHAPTER XI

AFFECTIONS OF THE MYOCARDIUM

(Dilatation, Hypertrophy, Myocardial Degeneration)

The evidences of myocardial affection are revealed by alterations in the size of the heart, irregular action, and inefficiency of the heart muscle. Since the maintenance of an efficient circulation depends on the integrity of the muscle wall, a knowledge of the state of the myocardium is essential to a due appreciation of the condition of any given heart.

UNFORTUNATELY, most of the changes that occur in the myocardium show no sign or symptom by which they can be recognised. Nevertheless, there are a number of manifestations which can be referred to the myocardium, such as various irregularities of the heart's action, changes in the size of the organ, and, it may be, in the character of the sounds. While, therefore, there may be no direct evidence of the changes, indirect evidence may be obtained from the study of the heart's efficiency. In many cases this study affords information of the highest value, so much so, indeed, that no examination of a patient should be concluded till a thorough understanding of the degree of efficiency of his heart muscle has been arrived at.

In discussing the affections of the myocardium dilatation and hypertrophy will be dealt with first. Later irregular action of the heart will be considered. The more prominent signs of inefficiency have been dealt with in speaking of heart failure.

Dilatation of the Heart.

The real meaning of dilatation of the heart has, so far, eluded recognition.

The factors producing this condition are still so obscure that still there remains much room for speculation. Like all other obscurities in medicine, the mystery surrounding dilatation provokes in the mind a sense of fear. A more intimate knowledge of the condition, however, though it has failed to afford full understanding, has yet shown that its significance varies greatly. The latest researches by Starling show that a dilatation of any of the chambers of the heart is produced when there is an increase in the pressure during diastole—the increase of pressure being due to a larger influx of blood. Starling shows that, when the heart is stimulated to greater effort, it dilates slightly, a slight increase in size being the best condition of the heart for increased effort. When the full effort which the heart can make does not quite overcome the resistance to the outflow, then the ventricle is not completely emptied, and so the residual blood added to the inflow tends to produce further dilatation. In other words, when failure of the heart sets in, the chambers do not completely get rid of their contents. These leavings are added to the new blood, and so the trouble goes on.

A certain amount of clinical evidence might be adduced in support of this contention. Healthy hearts exposed to severe bodily labour may dilate without impairment of functional efficiency. Chambers of the heart obstructed in their work—for example, by narrowing of the exit—tend to dilate, as in the case of the left auricle in mitral stenosis. Dilatation is common in various conditions that produce heart failure of a moderate as well as of a serious kind.

On the other hand, there are features which lead to the conclusion that the whole truth is not contained in this explanation. People suffer from heart failure, and

even die from it, in whom there is no dilatation. A man may suffer prolonged severe exertion and his heart may be exhausted without any sign of dilatation manifesting itself. In cases where the heart suddenly takes on an abnormal rhythm with marked impairment of its efficiency we may find that it dilates rapidly to a remarkable extent, but, in other cases, we may find no dilatation whatever. In certain febrile cases the dilatation of the heart is extreme, in other similar cases, with higher temperatures, there is no dilatation.

From such considerations as these we may conclude that there are other factors than dilatation, the result of increased pressure, which induce heart failure. Here indeed, as in all investigations dealing with vital functions, we find that the mechanical factor is not the only one.

A muscle fibre, when not in actual contraction, is not relaxed to its fullest extent. It is not limp like a cotton thread or other non-living structure. It still maintains a degree of contraction which offers a resistance—it may be a slight resistance—to extreme extension. This property of “tone” is capable of being influenced by certain agents. Thus, Gaskell describes a diminution of the degree of relaxation under the influence of certain agents as *antiarin*, and an increase in the degree of relaxation with other agents as *muscarine*. The possession of this property of tone by the muscle and its susceptibility to various stimuli in a positive and in a negative sense would account for some of the variations in the size of the heart to which I have referred.

Dilatation of the heart is recognised by an increase in the size of the organ, the borders extending beyond that area which we commonly assume a healthy heart will occupy. The most common sign of dilatation is an extension of the apex-beat or the left edge of cardiac dullness to or beyond the nipple line. We may get, in advanced cases, a descent of the apex-beat to the sixth

or seventh interspace and an extension of the dullness to the right beyond the edge of the sternum with increased epigastric pulsation. The extended area of dullness to the left, which is so frequently an evidence of dilatation, gives no clue to the chambers which are affected. Post-mortem examination will often reveal the fact of one or more chambers being much more dilated than the others. During life we can rarely tell which chambers are dilated. When there is extreme extension of dullness to the right, we may assume that the right auricle is greatly dilated, and when there is marked pulsation in the epigastrium, with indrawing during systole, we can assume that the right ventricle is dilated. The difficulty in recognising the dilated chamber is due partly to the fact that the pericardial sac on the right side is more *firmly fixed* to the superior and inferior cavæ than the looser investment on the left, so that any pressure tends to push the heart in the direction of least resistance. In carefully following a number of cases I have been able to observe a well-marked apex-beat, due to the left ventricle—recognised by the systolic out-thrust—replaced by a diffuse apex-beat, with a systolic indrawing—due to the great dilatation of the right ventricle. With subsidence of the dilatation the out-thrust of the left ventricle again became evident.

When we perceive an increase in the size of the heart in an individual going about in the enjoyment of health, we must bear in mind that dilatation of the heart may be a physiological process, and that it may be due to causes which do not hamper the heart in its work. It is mostly in the young that we will find dilatation to be seemingly a physiological condition. I remember being called, a great many years ago, to see a boy of ten who had fainted. I found the heart dullness extending well to the right of the middle line and half an inch beyond the left nipple line. At that time I was beginning to doubt whether such signs were evidences of a morbid condition, so I did nothing for

the heart, but allowed the lad to run about and play as if nothing had happened. I watched him and nothing did happen, and he has grown up to healthy and vigorous manhood with no sign of cardiac inefficiency. I have since then taken particular note of the heart in the healthy young, and have been struck by the frequency with which variation in size occurs. The subsequent histories of these cases of mine have shown that this sign does not indicate any impairment. Therefore, in examining the young, one must beware of attaching too much importance to the mere presence of a degree of dilatation of the heart, and of becoming obsessed by an unreasoning fear of the so-called over-strained heart or the athletic heart. In all cases in which dilatation is present, the functional efficiency of the heart must be inquired into, and if that has been good up to the time when some incident, such as syncope, has occurred, it may be taken for granted that the syncope was not primarily cardiac and that the dilatation had no part in the matter.

In people of middle life and later life changes of an obscure kind often take place which lead to an increase in the size of the heart. These are usually accompanied by a slight limitation of the field of response; but so long as the limitation is confined to what common sense indicates is in accord with the age of the individual, no importance need be attached to it. I remember, in my first years of practice, rejecting for life insurance a big, stoutish man, sixty-two years of age, because his apex-beat was one inch outside the left nipple line. He lived an active life for many years and died at the age of eighty-nine. As years went by I kept wondering at his activity, and I kept note of other similar cases and found that many middle-aged people had a distinct increase in the size of their hearts and yet led active lives with no sign of heart failure.

The causes of these increases I could never make out. There was no kidney trouble. The patients were

temperate-living men, and the heart's efficiency was good. I feel confident that an increase of the heart's dullness to a little beyond the nipple line, even when accompanied by murmurs and irregularities, such as the extra systole, has no serious significance if at the same time there be a good response to effort.

Heart failure, even to an extreme degree, may occur without dilatation.

There is a widespread impression, prevalent even among those who have devoted much time, clinically and experimentally, to the study of the subject, that when the heart begins to fail it dilates and that dilatation is, of itself, an evidence of heart failure. When, however, as I have already stated, the matter is critically examined, abundant evidence will be found to show that heart failure may end in death without any sign of dilatation having been perceptible during life and without the smallest evidence of dilatation being forthcoming on post-mortem examination. Moreover, many hearts, markedly dilated, show an extraordinary degree of efficiency. I have known a man with mitral stenosis and a considerable dilatation of the heart pursue his work as a mechanic, swing his forehammer and do all the heavy labour of his calling, with comfort and ease for over ten years. From such considerations we must conclude that dilatation, of itself, is not necessarily a phenomenon of heart failure, and that, further, it is by no means to be regarded as the only, the chief, or even an invariable causative factor in heart failure. Where dilatation has occurred and proceeded *pari passu* with heart failure there were other factors at work than dilatation. Thus I have seen cases of paroxysmal tachycardia, due to auricular fibrillation, suffer greatly from heart failure. With the onset of the attack the patient would become very short of breath, his face, in a short time, would become livid, his lips blue, while his liver

would enlarge and pulsate, and at the same time his heart would rapidly dilate, so that in a few hours the transverse cardiac dullness would have increased by over two inches. With the sudden resumption of the normal rhythm all these signs of cardiac failure would quickly disappear, and in a few hours the heart's size would markedly diminish. In these cases the heart failure was not due entirely, if at all, to the dilatation. In them the ventricular rate is so rapid that exhaustion of the heart muscle ensues, while the output of the ventricle is greatly diminished. The heart failure is thus independent of the dilatation. This view is supported by the study of numerous cases of permanent auricular fibrillation, and when dealing with that subject I shall refer again to the question of dilatation. Here I will anticipate merely by pointing out that we repeatedly meet with cases of extreme heart failure and great dilatation of the heart, in which, under suitable treatment, a marked improvement takes place without perceptible diminution of the size of the heart.

Dilatation is probably never produced by overstrain alone.

That dilatation can be produced in a healthy heart as a result of over-strain, more particularly among the young and athletic, is a belief widely held. I am convinced that it is a view which is not justified. For more than a quarter of a century I was engaged in general practice and had large numbers of young people under my care who played games strenuously and were employed in laborious occupations, and during that time I never saw a single individual who suffered from heart dilatation as the result of over-exertion. I think I may fairly say that this was not due to inability on my own part to recognise "overstrain." When one is a family physician watching individuals for years, the results of one's neglect are revealed in course of time.

During the years I was in London I had brought to me great numbers of the young of both sexes with

hearts said to have been impaired by overstrain. I did not find a single case of dilatation with heart failure among them. In the vast majority of instances the symptoms which were supposed to be signs of overstrain were purely physiological—for example, a slight increase in the size of the heart, irregular action, and systolic murmurs.

I do not assert that in certain circumstances dilatation may not be produced by over-exertion, but when this does occur the circumstances are wholly exceptional and very special. During the war I had opportunities of seeing a great number of cases of heart trouble which were supposed to be due to overstrain, but in each case there were present evidences of causative agents other than overstrain. Thus, one officer, who was in the retreat from Mons, complained of easily-produced exhaustion and breathlessness, and his heart's apex was one inch outside the nipple line. His story was that, during the fourteen days before the battle, he had had little sleep, not more than two hours in the twenty-four, on an average. In the retreat he was three days and nights without sleep, and was nearly always on the run. Finally he fell down unconscious. He was picked up, and, staggering along, fell again unconscious. An experience such as this is obviously a different thing from a game of football.

In other cases I got a history of some febrile illness while the patient was in the trenches, and he either did not go off duty or stopped off only for a very short period. With the resumption of his strenuous duties he gradually became more and more exhausted until he collapsed. In some of these soldiers there is a slight dilatation of the heart.

The dilatation which occurs in healthy hearts exposed to a strain is probably not due to "strain" alone, in the sense that the heart is being forced to do an excessive amount of work, but to other agents which depress the functions of the heart muscle, as, for example, the toxic

products resulting from an infection (see page 149). When one meets with dilatation as the result of excessive work, one finds, on inquiry, that the heart had already been damaged. This is seen in valvular disease (the valve lesion offering an impediment to the contracting chamber), or when there is some actual disease of the myocardium itself; or, again, when the two conditions are combined.

Dropsy due to heart failure is probably never present without dilatation.

The cardiac inefficiency that may accompany dilatation of the heart is not necessarily produced by the dilatation. In many cases, as in auricular fibrillation and auricular flutter, the more urgent symptoms arise in consequence of the increased rate which exhausts the contractile force of the chambers and diminishes the output. I am thus of opinion that there is no characteristic and invariable sign by which we can assure ourselves that any particular case of heart failure is due to dilatation. I have already called attention to the fact that we do not get dropsy without dilatation of the heart (see page 13).

Dilatation and digitalis and other forms of treatment.

With the cessation and removal of a provoking agent, as an attack of paroxysmal tachycardia or an infection, the dilated heart may return to normal limits as the result of rest and cessation of effort, and a damaged heart, dilated by overwork, may likewise diminish. But, beyond this, there is a notion held by many people that drugs, and other forms of treatment, can reduce the size of the heart. Among drugs those of the digitalis group have the greatest reputation. I must confess, however, that, though I have seen marvellous improvement take place in a great number of cases from the use of this drug, I have not been able to convince myself that it

has materially reduced the size of the heart. In many cases, indeed, where there has been marked improvement the size of the heart has not diminished, while, in a few cases where the size has diminished, I could not be sure whether the improvement was directly due to the drug or to the fact that the slower and more efficient beats emptied the heart more thoroughly.

In certain quarters, not famous for fullness of knowledge or for accuracy of observations, marvellous claims are made on behalf of certain methods of treatment—for example, exercises and baths. One meets with statements about the reduction of the size of the heart as the result of some movements or baths which, if true, would be nothing short of a miracle. These statements are sometimes accompanied by outlines supposed to represent the size of the heart before and after treatment. To the experienced physician, such outlines are so absurd as to deserve no notice. Nevertheless, one sometimes meets with them in the pages of books which make some pretence to scientific accuracy. Personally, I look upon these outlines and figures as nothing more or less than fakes. I have repeatedly had patients brought to me who were about to undergo one or other of these special forms of treatment. A diagnosis of enormous dilatation had usually been made; but when I examined the patient I found that the heart was perfectly normal in size. I have repeatedly seen individuals with hearts of normal size sent to places like Nauheim for treatment where they were told the heart was greatly dilated. The dilatation, they were subsequently informed, had been removed by the baths and exercises employed. I mention this as a caution against these and all other forms of cure over which there is a trail of commercialism.

Dilatation must be valued along with other evidence.

To sum up—having detected the presence of dilatation, it does not follow that there is necessarily much amiss,

nor that heart failure, if present, is due to the dilatation. In every case the functional efficiency of the heart must be taken into account; if that is in accordance with the health and age of the individual the dilatation may be ignored. If there be distinct limitation of the heart's power, then search must be made for a cause other than the dilatation that has induced this limitation. Our view of the patient's future does not depend upon the dilated heart as such, but upon the possibility of the removal of this other factor. If the cause cannot be removed, as, for instance, when there is degenerative change in the heart muscle, then the power of the heart to maintain an efficient circulation is the only safe guide.

Hypertrophy.

It is often impossible to recognise which chamber of the heart is hypertrophied.

In describing an increase in the size of the heart I have assumed that the increase is due to dilatation of one or more chambers. We find, however, though not generally, that certain chambers of the heart may be hypertrophied as well as dilated, or one chamber only may be hypertrophied while the others are dilated without hypertrophy. This is so well known that I need scarcely insist upon it; in mitral stenosis, for instance, there may be hypertrophy of the left auricle and right ventricle at first, followed later by dilatation of these chambers; later still great dilatation of both auricles may occur without hypertrophy.

The signs of hypertrophy are often rather indefinite. A powerful thrust of the apex-beat may be taken as representing one of them, and as indicating a hypertrophied left ventricle. But milder degrees of hypertrophy are not easy to differentiate from an excited heart action. Still, an apex-beat in, or just outside, the nipple line with a forcible protrusion during systole may be taken as evidence of moderate hypertrophy. A

powerful impact or shock inside the apex-beat and forcible beating in the epigastrium may be taken as suggestive of hypertrophy of the right ventricle. We have no evidence of hypertrophy of the left auricle, unless, indeed, the loudness and roughness of the pre-systolic murmur in mitral stenosis be regarded as such evidence. That it should be so regarded is, however, not certain, because a slight force is capable of producing a rough murmur, as is shown by the diastolic murmur of mitral stenosis. We have no direct evidence of hypertrophy of the right auricle, though in rare cases it can be inferred from the forcible waves sent back by the auricular systole into the jugular veins and the liver.

When hypertrophy is physiological in origin it is very moderate in extent.

The cause of hypertrophy is invariably an increased opposition to the emptying of the chamber. The systematic exercise of the heart's action keeps the organ in a healthy condition. A cessation of systematic exercise tends to diminish the vigour of the organ and even to produce atrophy of its active tissues. An increase in the exercise tends to produce an increase in vigour and an increase in the active tissues, so that, within certain limits, we may speak of a physiological hypertrophy, meaning thereby a state comparable to the large muscles of a blacksmith's arm. In such cases the hypertrophy is moderate in degree. We can no more get an abnormal hypertrophy of the heart than we can get an abnormal expansion of the biceps as the result of exercise. I make this remark because there are certain terms, often used, which are misleading, such as that of "athlete's heart." This term, I understand, is intended to denote an abnormally hypertrophied organ. Personally, I have never seen such a thing; and when my opportunities of observation are considered, I think it will be allowed that I would have seen it if it had

really existed. I have, on the other hand, seen many cases that were called "athlete's heart," but a careful examination of these invariably revealed other conditions which had been erroneously attributed to the athletics.

Morbid Hypertrophy.

Setting aside, then, the hypertrophy which is physiological, I am of opinion that all other forms of hypertrophy invariably occur as a consequence of some morbid condition obstructing the work of a chamber of the heart. The most extreme forms of hypertrophy are found with aortic regurgitation and adherent pericardium. There is something peculiar in the hypertrophy of aortic regurgitation, for though the muscle wall undoubtedly increases greatly in thickness, it is not at all certain that the hypertrophy is an increase of healthy muscular tissue. In aortic regurgitation, it is manifestly not the obstruction to the output alone which causes the hypertrophy, for in aortic stenosis with no regurgitation, or with only a slight amount of regurgitation, we never see the same degree of hypertrophy. It is possible that the dynamic effect of the regurgitating blood may participate in the production of this hypertrophy, yet, so far as I have looked into the subject, I have not found any constant correspondence between the degree of hypertrophy and the extent of the aortic leak. Moreover, the muscle itself is often so manifestly inefficient that one cannot but suspect that the hypertrophy is not genuine, even though the microscope reveals nothing but apparently normal muscular fibre. Indeed, the whole problem of hypertrophy is still very obscure. At present we are far from understanding the real nature of the condition, and the most we can do is to begin by observing carefully the facts as they present themselves, as an essential preliminary to the elucidation of the problem.

The same unsatisfactory conclusion is reached in

regard to the hypertrophy of adherent pericardium. An adherent pericardium does not necessarily cause hypertrophy, because one often finds an adherent pericardium on the post-mortem table when there is little or no increase in the size of the heart. On the other hand, if the pericardium is more or less fixed to the chest wall, in front to the ribs and behind to the spinal column—*e. g.* in adhesive mediastinitis—the hypertrophy may be enormous. To a certain extent this hypertrophy can be attributed to the fact that the muscle of the ventricle has to drag in the ribs during systole; but I have found remarkable hypertrophy in cases of adherent pericardium of rheumatic origin when there was no attachment to the chest wall. In these cases, I fancy, there has been some myocardial condition favouring an increase in the muscle wall, but I have not been able to demonstrate it.

Very big left ventricles are often found in kidney disease and in cases with persistent high blood pressure. In these cases, no doubt, the arterial resistance is an agent in the production of the hypertrophy; yet it is not clear that it is the only agent. The conditions that affect the peripheral blood vessels affect also those of the heart itself, and thus, it may be, damage the muscle fibres. From this point of view the post-mortem appearance of a number of my cases is significant. These hearts have shown, towards the apex, large areas containing but few muscle fibres and a great amount of fibrous tissue. The muscle must in these have been seriously impaired years before the final breakdown.

Compensation and the significance of hypertrophy.

With regard to the significance of special hypertrophies these must be considered along with the lesion producing them as in valvular diseases. Here I want merely to point out that hypertrophy, due to any morbid cause, invariably indicates an inefficient heart. This is of fundamental importance, as there has come

into use a term which is often used in a manner distinctly misleading. I refer to the term "compensation." The use of this term is characteristic of much that is vague and unscientific in medicine. Even those who use it never explain what they mean. Thus, we will find an individual with a murmur described as having a "mitral leak with good compensation," although his heart is perfectly normal and physiological in every respect. A patient with orthopnœa and dropsy from heart failure is spoken of as being in a state of "decompensation," and when the dropsy has disappeared and the patient can lie down, "compensation" is said "to be restored." I have even known a distinguished professor of medicine rejoice the heart of a man with aortic disease, who could not walk one hundred yards without being pulled up by pain, by telling him that his outlook was good as there was no dropsy, and, therefore, compensation was good! Yet this man died shortly after from heart failure, and no dropsy ever appeared.

If a patient has hypertrophy which is not physiological his power of response is limited; if this fact be recognised when there is still a fair response to moderate effort and the individual be instructed as to his limitation and told only to take such exercise as gives him no distress, a fair prospect presents itself. The trouble is that these patients think that their limitations correspond to those of healthy people, and so try to lead the lives of healthy people, with the result that, instead of recognising the sensations indicating their limitations, they put them aside, too often with disastrous results.

Myocardial Degeneration.

It is not necessary to point out that with advancing years changes occur in the myocardium as in all other tissues, impairing the heart's efficiency. These changes have probably been set up in early or middle life by acute affections (as rheumatic fever) which have given rise to no physical sign. It is possible that, as extra

systoles become more frequent in the later years of life, they are associated with some obscure changes in the heart muscle. Apart from these phenomena there is usually no physical sign of myocardial change beyond an occasional increase in the size of the heart; the most striking—often, indeed, the only—evidence is the limited response to effort.

There is a group of cases—patients who begin to exhibit their symptoms just before, at, or just after, sixty years of age—in which the symptoms are so marked and so manifestly the outcome of myocardial degeneration that they deserve special attention. The most typical cases of this group are those with chronic Bright's disease when heart failure is the cause of the more prominent symptoms. Dyspnoea, in these cases, is readily provoked by effort, and Cheyne-Stokes respiration and attacks of cardiac asthma are not infrequent. Pain is sometimes provoked by exertion, and in the later stages the attacks become typical of angina pectoris.

Myocardial exhaustion is accompanied by other signs. The arteries are often thickened and the blood pressure high; and the heart is enlarged—the apex being thrusting and forcible and situated outside the left nipple line. The heart's sounds are often muffled; sometimes there is a well-marked systolic murmur. The rhythm of the sounds is frequently of that triple character—"the gallop rhythm." The cause of this gallop rhythm seems to be a lack of synchronism in the contractions of the ventricles. In some of my cases Sir T. Lewis has demonstrated from electro-cardiographic records that there is a slight delay in the contraction of the right ventricle due to damage of the right branch of the auriculo-ventricular bundle. There is also sometimes a delay in the transmission of the stimulus from auricle to ventricle. The rhythm of the heart is often quite regular, but frequently we meet with extra systoles and the pulsus alternans. Dropsy gradually occurs and may become very extensive.

This description, though typical of chronic Bright's disease, is also characteristic of cases that have never shown albuminuria—though albuminuria may appear at their later stages.

These cases usually drift and die, treatment being of little benefit.

The post-mortem examination of some of these cases has revealed extensive fibrosis of the heart muscle.

CHAPTER XII

HEART IRREGULARITIES

The Meaning of Irregular Action of the Heart; The Youthful Type of Irregularity; The Adult Type of Irregularity (Extra Systoles); The Pulsus Alternans.

Irregular action of the heart is of very frequent occurrence and throws a flood of light upon the mechanism of the heart-beat.

IN my early days in practice, while carrying out close observations on many cases, I was struck with the frequency of irregular action of the heart at all periods of life. When I recollected what I had been taught concerning this condition I found that, apart from some vague incidental reference to it, I had never heard a word spoken which might direct attention to the different forms, their mode of production, and their significance. Even the text-books of the day forbore mentioning the matter, save for a passing reference to an "irregular" heart. The writings of the greatest masters slurred over the subject; and to many, even at this day, the whole accumulated knowledge possessed by the profession is expressed in Broadbent's book on "The Pulse," published in 1890. It might be worth while to read this book and compare the knowledge possessed by that able and distinguished clinician with the knowledge contained in some book written in recent years by a physician familiar with the present-day aspects of the subject. I have little hesitation in saying that in no other department of medicine has advance in general knowledge of an important subject been characterised by such wonderful strides.

The due appreciation of irregular action of the heart has thrown a flood of light on the physiology of the heart; brought before us, with precision, diseases of the heart the origin and manner of which had hitherto been shrouded in impenetrable darkness; given us a new conception of how drugs affect the heart beneficially, and enabled us to administer drugs with a scientific precision undreamed of before. The differentiation of irregular actions into groups, based on physiological principles, has given us further the basis for a sound prognosis.

The real significance of irregular heart action has not been recognised.

Although so little has been said about irregularities by teachers and writers, the subject itself has by no means been ignored in practice. So ingrained had the belief become that a heart to be normal must be regular, that when an irregular heart was met with it was looked upon with suspicion. Many individuals, with perfectly sound hearts, have been rejected for life insurance and for appointments in military and civil life because of the presence of some innocuous irregularity, while tens of thousands have had their lives restricted and have been subjected to prolonged and useless treatment for a condition that called for none. On the other hand, cases, where the nature of the irregularity should have revealed the necessity for treatment, were not properly understood, and so were never properly dealt with.

This language may appear extravagant; but if one considers what, at this moment, the attitude of many a physician of good standing would be towards an individual with an irregular heart whom he was examining for life insurance, its applicability will be apparent. Would the physician accept, reject, or accept with an increase of premium? Strong statement as it may seem, I have a shrewd suspicion that, no matter which

course he took, he could not give rational grounds for his action. Not long ago an eminent physician stated that my investigations into the meaning of irregular action of the heart revealed nothing new; he and his colleagues understood perfectly well all the essential matters concerned with irregular action. Within a few weeks, as it happened, my opinion was asked regarding the irregular heart of a girl whom this physician had ordered to stay away from school for three months. The order had been given wholly on account of the irregular action. Yet, in my view, this condition, far from being a sign of heart trouble, was, in the girl's case, a sign that the heart was free from trouble. A short time ago I took part in a discussion on prognosis in heart affections at a Society of Life Insurance physicians. I found the idea prevalent that a man with an irregular heart should either be rejected or have his premium increased. No attempt was made to differentiate the types of irregularity; and the necessity of carrying out a differentiation, when it was insisted upon by me, was regarded sceptically by some speakers. In other words, it became clear that the element of pure guess-work was strongly prevalent. I put the matter in this way because I find that it is impossible for the average physician to realise how great an amount of information may be obtained from a thorough appreciation of irregular action of the heart.

To appreciate the meaning of heart irregularities, careful differentiation had first to be made, followed by prolonged observation of individuals.

As I wish to define clearly what research in medicine really means, or, at all events, to indicate what is my conception of its meaning, I will cite briefly the steps which led to my present knowledge of the subject of irregular action of the heart. I do this in order that the method by which I arrived at my conclusions, and

the grounds on which they are based, may be understood and appreciated.

In my early attempts to understand the reasons for my patients' complaints I endeavoured to unravel the meaning of such phenomena as I did not understand. Thus, one sign that I frequently met puzzled me, namely, the movements in the jugular veins. I was profoundly ignorant of the subject, but it was evident to me that no advance could be made until the waves in the vein were recorded in some manner or other. After much labour I devised a method by which the movements of the veins could be conveyed along a tube to a tambour. By attaching the tambour to a Dudgeon sphygmograph I was able to register the movements of the jugular vein on the same paper and at the same time as the sphygmograph lever recorded the radial pulse. In this simple manner I was able, by comparing the beats of the radial pulse with the movements of the vein, to place all these movements in their appropriate places in the cardiac cycle, and so distinguish the force that gave rise to any particular wave or depression. I was then able to detect the movements of the right side of the heart on a tracing which also gave the movements of the left ventricle (radial pulse).

While making the observations as to the nature of the jugular pulse I frequently came upon people with irregular hearts, and as these irregularities had for a long time been a puzzle to me, it struck me one day that I had better see what the nature of the movements in the jugular vein were at the time the heart was acting irregularly. I had no suspicion, when I did this, what the outcome of the investigation would be, but I soon found that it gave me a new and unexpected field for observation. I found, very soon, that in some irregularities all the chambers of the heart participated, that in others the auricles beat regularly while the ventricles acted irregularly, and that in others the auricles ceased to act. Again, I found that the auricles might act more

frequently than the ventricles and that the time between the contractions of the auricles and the contractions of the ventricles might be increased. Although these things are quite clear now, twenty-five years ago no one suspected them to exist. I remember very well the first occasion on which I detected an irregularity of the radial pulse and apex-beat while the auricular systole was perfectly regular—the first occasion, that is to say, on which the nature of a ventricular extra-systole was demonstrated in the human heart. Though now this fact is merged into everyday knowledge and the manner of its discovery has become of no importance whatever, I mention it because of the glow of delight which I felt on perceiving the nature of the irregularity; for it is by the lifting of the fringe of Nature's curtain that the inquirer feels so bountifully rewarded for endless trouble and toil.

Since that time many workers have laboured in this field and other methods have been employed. Many have found the irregular heart a fascinating and instructive study. But, notwithstanding its fascination, and notwithstanding the profound knowledge of the heart's action that has been acquired, the part of the work which has revealed the mechanism of irregular heart is but preliminary. The real work only begins after the physiology has been worked out, and the guide to the further research is found in the question: *What happens to the people who show these irregularities?*

The answering of this question necessitated a long and weary pursuit, a pursuit brightened, nevertheless, from time to time by the discovery of some new fact. Great numbers of patients had to be followed during many years, and close observations regarding the manner in which they bore the stress and strain of life, in acute illnesses and in laborious occupations, had to be made. The changes, too, that occurred in the heart itself as time went on had to be recognised. Large numbers of cases were essential to the work, for by this means only

could one find the age incidence of the different forms of irregularity and thus determine whether or not antecedent illnesses, such as rheumatic fever, had any bearing on the causation of any particular irregularity.

The great majority of irregular actions of the heart are of two kinds, one which occurs in youth, and the other which occurs in adult life.

Proceeding on these lines, after I had collected over a thousand cases I sought for some definite basis of classification. With the assistance of the jugular pulse I was able to recognise different forms according to the mechanism of their production. Two large groups, which included, between them, about 90 per cent. of all the cases, emerged distinctly. These two groups differed. In one all the chambers of the heart participated in the irregularity, the contraction in each case being normal, while, in the other, the ventricle contracted prematurely while the auricle maintained its rhythm, or both auricle and ventricle contracted prematurely—the irregularity which is now called the ventricular and auricular extra-systole.

I was also able to separate the two groups by a second method, namely, the age incidence. I found that the former group occurred predominantly in the young (“the youthful type of irregularity”) and that the latter group occurred predominantly in the later decades of life (“the adult type of irregularity”).

This classification into youthful and adult types does not find favour with the great majority of writers, who prefer to recognise the groups by the names which indicate their mechanism. As a rule, it is a matter of indifference what name is used so long as the name is understood; the reason I am about to employ this clinical nomenclature, instead of the physiological one, is that the names—youthful type and adult type—carry a meaning which is of great value in practice.

The Youthful Type of Irregularity.

In the youthful type of irregularity all the chambers of the heart participate, and it is present in perfectly healthy hearts.

The individual showing this irregularity is never conscious of its presence. It can best be recognised by listening to the heart, as the ear can detect variations in the rhythm better than the finger. The heart will be found to vary in rate, a slight increase occurring as a rule during inspiration and a slowing during expiration (sometimes, therefore, called respiratory irregularity). Sometimes it is not possible to make out the relation to the respiration very distinctly, but if the

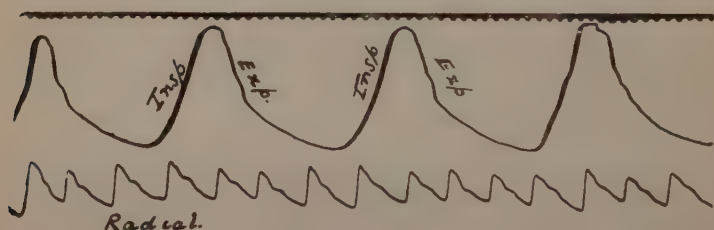


FIG. 9.

Youthful type of irregularity showing respiratory variations—diminished rate during expiration and an increased rate during inspiration—more marked with deep breathing. (See Fig. 10.)

patient breathes slowly and deeply it becomes easy of recognition. This should be borne in mind because the nature of an irregularity in the young is sometimes very puzzling and this simple procedure at once clears it up (Figs. 9 and 10). There are a few cases in which the heart slows for short periods, but in which its rhythm does not show this relation to respiration. The immediate cause of this irregularity is vagus stimulation—the act of breathing being sufficient to stimulate the vagus which, acting on the sino-auricular node, causes a transient slowing of the whole heart (hence also sometimes called vagus or sinus irregularity).

A slight form of this irregularity has been detected in infants. It becomes most marked at the periods of life when the heart-rate tends to fall from the rapid rate associated with infancy, *i. e.* between the ages of five and ten. It is often well marked about puberty and may last until thirty years of age or after. I have not followed cases with sufficient care to find out when it disappears, but it becomes rare towards thirty. Certain neurotic individuals may show it late in life. The period when it is most marked is after the subsidence of some

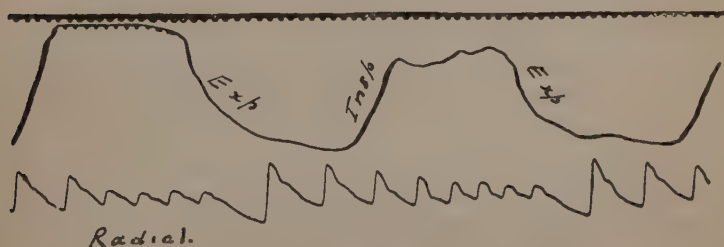


FIG. 10.

Continuation of Fig. 9 when the patient breathed deeply and slowly. The increase in rate during inspiration, and decrease during expiration, is well marked.

febrile condition, and an imperfect knowledge of this irregularity causes many physicians to imagine that it is a sign of gravity when it occurs after rheumatic fever, measles, diphtheria, or influenza. In some healthy young fellows it is very marked when they are at rest, and especially after some fatiguing exercise.

Considering its great frequency and the fact that it may be very well marked, it is astonishing that so little attention has been paid to it by physicians. In large text-books, even in those dealing with children's diseases, it may never be mentioned. In fact, the only article I have found dealing with it is one by Corrigan, written more than fifty years ago; in this article it is imperfectly described and looked upon as an evidence of impairment of the heart, if not of disease. When I have directed attention to this irregularity I have not

infrequently been told that everybody knows all about it; but I find from experience that most people look upon it as something wrong. The first consultation I had in London was on a young girl who showed this irregularity. She lived in the country, and one day she fainted. A physician from London was summoned to see her. He found her heart very irregular and he had her conveyed to London with the greatest care. She was put through a course of Nauheim baths and some form of exercises, but still the heart remained irregular. She was then put through another course, with no better result. When I saw her she had been three months in bed. Yet her heart was perfectly sound and had only this well-marked youthful type of irregularity. It was this irregularity which had frightened the physician. Some six years ago a lad of sixteen was brought to me because he was supposed to have an incurable affection of the heart. Two years before I saw him he had had a febrile attack while at a public school, and after the fever subsided his heart was found to be irregular. He was treated at school for a period with no success. He was then removed to London and again treated for some months. He was next taken to Paris to a reputed authority and treated by that authority, but with no success. Finally, he was taken to another place on the Continent where was a physician famed for his cures by injections of sparteine and strychnine and other drugs. Still no result followed, and the physician informed the sorrowing parents that the heart was permanently and incurably affected and that the boy must lead a very quiet life, play no games, and avoid all violent exertion. He had been leading this miserable existence for a year before I saw him, yet when he was brought to me all that was present was a very well-marked youthful irregularity. The boy was told at once to begin leading the life of a healthy youth. He did so, and is to-day a stalwart, vigorous, healthy young man.

It will be evident from this that I look upon the youthful irregularity as a physiological sign, and not as evidence of impairment of the heart. Far less do I look upon it as evidence of disease of the heart. The observations on which my view is based extended over a great number of cases. Having detected the irregularity in young men I have submitted them to no treatment. Yet they indulged in vigorous games and afterwards led laborious lives or engaged in occupations needing the exercise of great muscular strength. I have seen girls, in whom I detected the irregularity, grow up, marry, and have children. In not one of these cases have I ever seen the slightest sign of heart failure.

The presence of the youthful type of irregularity after a febrile illness may be taken as an indication that the heart has escaped infection.

There is another aspect of this irregularity to which I wish particularly to direct attention, as it raises a question that I have been unable definitely to answer. As I have said, the irregularity appears most markedly after a febrile attack, while, on the other hand, any circumstance which increases the rate of the heart causes it to disappear. After an acute febrile attack, such as rheumatic fever, there may be some doubt as to whether the heart has escaped infection. It occurred to me many years ago to inquire whether the presence of this irregularity might not be taken as an evidence that the heart had escaped infection. The disappearance of the irregularity, with increase in rate by fever, showed that the weak stimulus from the vagus was ineffective during the time that the febrile process was present; but that the vagus stimulation reasserted its sway when this factor was removed. I reasoned that the presence of an active process in the heart, such as that occurring in acute rheumatism, would prevent the appearance of this irregularity presumably by some disturbance of the

normal reflexes, and hence that the reappearance of the irregularity must indicate that the heart had become free from the irritating rheumatic poison or infection. I have, as I have stated, been putting my theory to the test, and so far I have not come across a single exception. Bear in mind, however, that I only put this theory forward in respect of cases where the irregularity is well marked, and where, at the same time, the pulse-rate has fallen below 70 or 80. Let me add that even when there has been present a distinct systolic murmur, which may have appeared during the febrile attack, I have taken the irregularity as an evidence that the heart muscle has escaped.

A few years ago I was asked to see a lad who had just recovered from a mild attack of rheumatic fever. While the patient was still in bed his doctor recognised the onset of a marked irregular action of the heart. There was present also a soft systolic murmur. The doctor feared that an endocarditis was present, and a consulting physician, who was called in, concurred in this fear. The lad was therefore ordered to be kept on his back for from six weeks to two months. A week later I was asked to see him and I found him feeling well. His temperature was normal, his heart-rate about 70, the rhythm showed a characteristic irregularity of the youthful type; the heart itself was normal in size, and a soft systolic murmur was audible. I said that the heart had escaped all damage and that there was no endocarditis. I advised that the patient should get up. The result justified this view; the lad is now well, strong, and fit.

The irregularity of the youthful type may be present in old valvular affections and can then be accepted as an indication that there is no active process going on in the heart.

The Adult Type of Irregularity (the Extra-Systole).

The extra-systole is rare in the young, but increases in frequency with advancing years.

I had no idea of the frequency of what is called the intermittent pulse until I began to search systematically for it. I found, on taking tracings as opportunity offered, that this irregularity was present in 50 per cent. of perfectly healthy, pregnant women. I next considered the age incidence and found that the condition was infrequent before puberty, more frequent between thirty and forty, and that it occurred with increasing

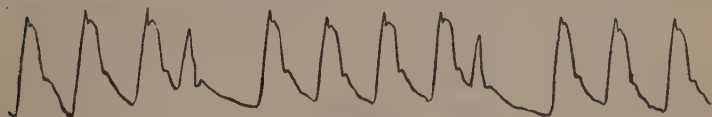


FIG. 11.

The smaller beats are premature and due to extra-systoles.

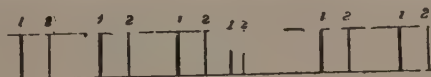


FIG. 12.

Diagram representing the sounds of the heart with an occasional extra-systole—two short, sharp sounds occurring prematurely and followed by a pause longer than the normal interval between the second and first sounds. The downstrokes 1, 2 represent the first and second sounds of the heart.

frequency from the latter age until after sixty. Practically every healthy individual over sixty whom I had the opportunity of examining at frequent intervals had it. It was for this reason that I called it the adult type of irregularity.

The recognition of this irregularity is very easy in the majority of cases. There is a small premature beat of the ventricle which is not always perceptible to the finger and which thus gives rise to the impression that the heart is intermittent (Fig. 11). If, however, the heart be auscultated, two short, sharp sounds will be heard during the early portion of the pause (Fig. 12).

These sounds are produced by the premature beat or extra-systole. In some cases there can only be detected one low muffled sound, and in a few cases no sound can be detected at all. In these latter there may be some difficulty in differentiating the extra-systole from the dropped beat of heart-block, but the latter condition is rare and is usually associated with manifest signs of heart weakness, whereas the extra-systole is not necessarily so associated, and tends to disappear when the pulse rate is increased, *e. g.* after exercise.

The patient's sensations are important for the diagnosis.

The patient's sensations are often a very good guide, and they may vary very much. Some patients are quite unaware of the irregular action, others are conscious of the pause, while others are distressed by the big beat that follows the pause. Some have a transient sensation of fullness in the region of the heart, while others have a suffocating feeling in the throat and may give a short cough. It is necessary to make oneself familiar with these sensations, because in many cases the irregularity is not present at the time the patient is under examination. Moreover, the consciousness of the presence of the sensations often fills the patient with fear, and therefore it is necessary to understand the causes of these terrifying feelings. The feelings may come on only at infrequent intervals, as, for example, after a full meal; or they may occur in the night, or they may be present at irregular, short intervals. Often they occur regularly after one (pulsus bi-geminus, Fig. 13), two, three or four normal beats. The peculiar sounds, while the pulsus bi-geminus is present, are shown in Fig. 14.

Apart from the worry and fear which it occasions, the extra-systole is of little significance.

The real matter which concerns us as clinicians is the effect which this irregularity has on the patient's mind. The peculiar sensations connected with it, such as consciousness of the lengthened pause after the premature ventricular contraction, give the victim the idea that his heart may stop altogether, while the big beat worries him, especially in the night. His anxiety may be added to by the doctor's uncertainty and by his failure to cure the trouble. A friend of mine, whose wife was being treated at Nauheim, was once much upset at that place by being told that his heart was "affected"



FIG. 13.

Pulsus bi-geminus due to an extra-systole occurring after each normal beat.

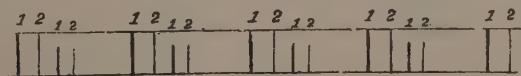


FIG. 14.

Diagram representing the sounds of the heart in a case of pulsus bi-geminus as in Fig. 13.

when he had casually suggested that he should have one of the baths himself and had submitted to examination as a preliminary. The doctor felt his pulse, assumed an air of great gravity, declared that the heart was "bad," and insisted on his taking a "cure." When my friend returned home I had some little difficulty in restoring his confidence in himself, for the treatment had had no effect on the irregularity, and he kept feeling his pulse for the intermission. That is nearly twenty years ago, and he is quite well to-day, although the pulse still shows these intermissions at times. Fear of this kind will cause strong, healthy men to put themselves to any amount of trouble in order to "get cured."

A man consulted me some years ago because of an intermittent heart. When he sat down I noticed that his right hand instinctively sought his left wrist and that a finger was kept on his pulse. I knew at once what I had to deal with. His story was this: His doctor, in one of the Western States of America, had found his heart irregular, and, being unable to cure him, had sent him to a celebrated physician who had treated him for some weeks with no improvement. He next went to New York and was again treated by a distinguished physician without result; then he was recommended to go to Nauheim. On his way through London he consulted me, and, on inquiry, I found that his whole trouble was due to the consciousness of these extra-systoles. I asked him when he could get a ship back to America, and he said in four days. I told him to take it, get back to his work, and never feel his pulse again. This he did, and I heard from him lately to the effect that he was in good health. When he had too much office work he was still conscious of the intermissions, but when he took an active holiday in the woods, hunting and shooting, the intermissions disappeared.

While some people have these extra-systoles permanently during many years, most people have them only at intervals; in some cases circumstances can be found which provoke the extra-systoles—for example, indulgence in tobacco or alcohol. Certain articles of food such as tea and coffee, may provoke them, while mental worry and sleeplessness have the same effect. On the removal of these provoking causes the extra-systoles may disappear, but they always tend to return, and in the later years of life they become more and more persistent.

In many people, in whom extra-systoles are constantly present, they disappear during a febrile illness.

Prognosis.

Seeing that healthy men and women may present this form of irregularity, it can be accepted that extra-systoles, in themselves, are not indications of disease or of impairment of the heart's efficiency. When there are signs of heart failure the prognosis should be based upon the other symptoms present and not on the presence of the extra-systole.

It may therefore be stated that, when the extra-systole is the only abnormal sign, the prognosis is good, and when it is associated with other signs the prognosis should be based on these other signs.

Where I have not yet been able to see my way clearly is in regard to the meaning of extra-systoles in acute affections. Thus, during an attack of rheumatic fever or shortly after the subsidence of the fever, extra-systoles may appear. Whether they indicate that the disease has invaded the muscle I do not know, as I have not been able to follow up a sufficient number of cases to comprehend their relation clearly. In a few cases their presence was coincidental with an affection of the heart. Similarly, in acute affections, such as pneumonia, their sudden appearance has frequently been heralded as indicating a very grave condition; but John Hay says he has noted their appearance in pneumonia without any dangerous result.

The Pulsus Alternans.

Of the rarer forms of irregularity the pulsus alternans stands out because of its prognostic significance. It is rarely possible to detect it by the finger or by auscultation, but its presence is revealed by graphic records of the radial pulse. The fact that the force of the larger beats in the arteries is greater than the smaller beats affords us an opportunity of detecting the presence of the pulsus alternans by a simple method. On page 163

I describe how to take the blood pressure by the auscultatory method, where the armlet is distended until the artery is obliterated. On listening with the stethoscope at the bend of the elbow, when the pressure in the bag is gradually released, the returning blood wave in the artery under the stethoscope shows its presence by a sound. When the *pulsus alternans* is present this sound is only half the rate of the heart at first, but when the pressure falls another five or ten mm. Hg., the weaker beat comes through, and one can detect then a sound accompanying each beat of the heart. At first the beats will be unequal in volume, but as the pressure falls lower they become equal. The *pulsus alternans* can be distinguished from the *pulsus bigeminus*, where the smaller beat is due to extra-systoles, by the fact that, when all the beats are heard, the sounds are perfectly regular in the case of the *pulsus alternans*, whereas there is always a delay after the weaker beat in the *pulsus bigeminus*, agreeing thus with the characteristic features of Figs. 13 and 15.

As a rule, when the *pulsus alternans* is present, there are no changes in the sounds of the heart, but occasionally I have found, when there was a musical murmur present at the aortic area, that this musical sound varied in intensity with the large and small beats.

The pulse beats are perfectly regular in rhythm, but the beats regularly vary in size—a big beat and a small one alternating (Fig. 15). Sometimes the variation in size is very slight and only brought out after an extra-systole. This appearance after an extra-systole is a very instructive feature, for in many cases there would be no evidence of this tendency otherwise.

There are two conditions in which this irregularity tends to appear: (1) during an attack of paroxysmal tachycardia (as auricular flutter); and (2) when there are degenerative changes in the heart muscle, producing exhaustion. It is in this latter condition that it is an evidence of gravity. There are always other evidences

present, the heart being generally increased in size, with a great limitation of its reserve capacity, as shown by dyspnœa on the slightest exertion, while there may be attacks of angina pectoris and cardiac asthma. Associated with these symptoms it invariably indicates an incurable exhaustion, and not infrequently it heralds a fatal termination within a short period. Occasionally it is detected before the exhaustion is so extreme, and, with rest, a certain degree of recovery may take place and the individual may lead a restricted life for an indefinite period.



FIG. 15.

Radial tracing showing the pulsus alternans. Each period is of exactly the same duration (six-tenths of a second). Compare with Fig. 13.

In paroxysmal tachycardia it is not of such grave significance. It is probably related in some way to the temporary exhaustion of the ventricular muscle from the long-continued increase in rate.

I have detected it in a few cases of pneumonia, and all proved fatal, but the number has been too few for me to say that it is always of such grave import.

On rare occasions the alternating beats have been noted during a temporary exhaustion of the heart, and even when there has been only slight heart failure, and it did not seem to have a grave significance. It is therefore of grave significance only when it is associated with other signs of exhaustion.

This abnormal rhythm must not be confused with the pulsus bi-geminus—which owes its peculiar rhythm to the alternation of a normal beat with an extra-systole (compare Fig. 13 and Fig. 15).

CHAPTER XIII

AURICULAR FIBRILLATION

The recognition of auricular fibrillation opens up a new era in the study of affections of the heart.

As years pass and my knowledge of affairs connected with the heart increases, I am more and more impressed with the great importance of the condition which, of late, has been termed auricular fibrillation. The value of the work devoted to the study of this condition does not consist merely in the ability we now possess to recognise it, but rather in the light thrown upon the heart's varied activity and upon its failure by the study of the life-history of our patients. The recognition of all the facts connected with auricular fibrillation brings into prominence numerous problems which have hitherto been ignored or only imperfectly recognised. Thus a new era in the study of heart affections is opened up. I state with firm conviction that no one can appreciate the essentials in the recognition of heart disease and heart failure until he has made himself familiar with the varied phases of this condition.

For the due appreciation of auricular fibrillation the watching of individual cases for many years is necessary.

Let me repeat what I have insisted on so frequently, that complete knowledge can never be acquired by the physician who limits his horizon to hospital practice and the consulting-room. Although it is but a few years since physicians began to recognise this condition, numerous articles have already been written on the subject with that assumption of complete knowledge which is so characteristic of much in medical writings.

This is to be deplored, because it is quite manifest that a subject of this kind can only be understood by means of the study of individual cases before the onset of fibrillation and also for a long period of years after its onset. Writers who have only been able to recognise the condition within the last few years cannot have had time to acquire more than an elementary and imperfect knowledge of the subject.

I am not saying this to depreciate the work of these writers, but to emphasise the fact that, in this most important subject, the methods of research which are peculiar to clinical medicine demand a totally different application than that usually employed by hospital and laboratory workers.

To the general practitioner especially, auricular fibrillation opens up a most fruitful field for study. By examining these cases carefully in the first instance and keeping in touch with them, as time goes on an insight into what heart failure means and how to combat it will be obtained that will lead directly to a fuller knowledge of the whole problem of heart-disease and its treatment.

The onset of heart failure by "back pressure" is shown by the recognition of auricular fibrillation to be misleading.

It is probable that from no other condition can such a true knowledge of what heart failure really means be obtained. This knowledge will speedily reveal how false is the doctrine that heart failure results from "back pressure." This doctrine, with its attendant verbiage of "compensation" and "decompensation," has blinded the profession to the real facts for more than sixty years. The usual explanation of heart failure is that it works backward from the point of origin. Thus a mitral or aortic leak embarrasses one chamber after another by "back pressure," and when at last the right auricle is embarrassed the heart takes that irregular action which is so frequent in heart failure

with dropsy. This view has not been derived from patient observation of individual cases, but has been evolved from speculation as to the cause of certain imperfectly understood symptoms.

The recognition of how auricular fibrillation embarrasses the heart in its work provides the key to prognosis and treatment.

The management of heart affections is guided by the knowledge acquired regarding the effect of this condition. One must differentiate clearly between what is of significance and what is non-essential. The treatment of a heart case does not mean merely the giving of a bottle of physic as a palliative measure, but requires careful study of the conditions present, so that changes, which foreshadow heart failure and which, if not guarded against, may end in serious failure of the heart, may be detected. The recognition of such changes makes it possible to plan out the patient's future, to place the treatment on a scientific basis, and, by avoiding unnecessary restrictions, to permit such liberty as shall enable the individual to lead a useful life.

The reasons for the beneficial effects of the digitalis group of drugs can now be explained.

An equally striking result which the recognition of auricular fibrillation has produced is the revelation of the effect of digitalis on the human heart. The light thrown on this part of the subject is indeed revolutionary, so much so, indeed, that the vast bulk of the literature on digitalis will have to be scrapped. Authorities have differed hitherto in regard to the dosage and the kind of preparation of the drug which should be employed. The conditions that seemed to call for administration were never clearly defined, so that it may be said that anything which seemed unusual in the heart, whether a physiological sign or a pathological, was taken as a valid reason for giving digitalis. The dangers attending the use of the drug have never been

clearly explained, but have been hinted at in oracular phrases which, while they alarm, do not instruct.

Now, the recognition of auricular fibrillation has shown that the heart while in this condition is peculiarly susceptible to digitalis, and that, in many cases in the past, the drug has been given and has acted most beneficially. But it was never realised that this peculiar reaction to the drug was an attribute of the abnormal heart action. Hitherto the reasoning has been that because, in certain cases, the rapid action speedily became slow under the influence of digitalis, therefore all rapid hearts would benefit by its administration. The recognition of auricular fibrillation has enabled us, on the contrary, to indicate with precision the hearts which will benefit by the drug and the hearts upon which the drug will have little or no effect.

We may state broadly that while the susceptibility of the heart to digitalis, in individuals with a normal action of the organ, may be extremely slight, as soon as auricular fibrillation occurs the susceptibility becomes at once very marked.

This peculiar and striking reaction to digitalis, exhibited by the heart when in a definitely marked pathological state, necessarily suggests a new conception of research in the matter of the action of drugs. It is manifest, for instance, that experiments on animals can only afford limited information in regard to this subject; for the reaction of a normally acting heart to this drug has been found to be fundamentally different from the reaction of a heart acting with an abnormal rhythm, such as auricular fibrillation. This line of thought opens up great possibilities.

The discovery of auricular fibrillation was the outcome of a long and patient research.

Though the term "auricular fibrillation" is of comparatively recent introduction all the essential clinical features of the condition were described long before

the actual name was applied. The recognition of the fact that the auricles fibrillate has not added anything to the clinical picture or assisted in its recognition, prognosis, or treatment.

It is now more than thirty years since I was first able to distinguish this condition as being one by itself. I was able to do this chiefly because cases of auricular fibrillation never show any waves due to the auricle in the venous pulse. Noting that the condition occurred most frequently in the elderly and in those with rheumatic hearts, I kept in touch with a large number of people in whom I saw reason to anticipate its occurrence. In 1897 a patient, whom I had been watching for a great many years, suddenly developed extreme heart failure with a rapid, irregular action of the heart. For years before this time I had been taking records and tracings and had found invariably, in tracings from the jugular and liver pulse of this particular patient, waves produced by the contracting auricle. Moreover, I had noticed that, in his case, there was always a presystolic murmur. After the irregular action appeared these auricular waves in the jugular and liver pulses and the presystolic murmur disappeared ! Thus, all the evidence of auricular activity had ceased. From careful consideration of numerous other cases I was able to confirm the observation, and for many years I speculated on what the auricle was doing after it ceased to appear in the tracings. Thus, in a book I published in 1902, I described the condition as "paralysis of the auricle," and later I described it as "nodal rhythm." When experimental proof was adduced, by Sir T. Lewis in 1910, that the cessation of auricular activity was due to the fibrillation of the auricle, I had no hesitation in accepting the term "auricular fibrillation." During the intervening years I had been collecting cases and following them up; I was therefore able to produce many facts on which to base the diagnosis, prognosis, and treatment of these cases, although, as indicated

above, the condition had been called by another name than auricular fibrillation. Some of my patients died shortly after the inception of the new rhythm, while others lived for months, and even for many years. It is clear, therefore, that an understanding of the effects of fibrillation cannot be acquired in a few months or even in a few years.

What is auricular fibrillation?

The term "fibrillation" is applied to a curious condition of the muscle fibres of the heart, in which the individual fibres, instead of contracting in an orderly and simultaneous manner during systole, contract rapidly and independently of one another. The auricle, when in a state of fibrillation, presents an entirely different aspect from that which it does during its normal action. It stands still; systole never takes place, and meanwhile the walls quiver with fibrillary contractions. A normally acting auricle can be thrown temporarily into auricular fibrillation by electrical stimulation.

If the ventricles pass into fibrillation death is instantaneous.

Auricular fibrillation is usually persistent, and the heart may act for indefinite periods.

In the majority of cases, when auricular fibrillation sets in, it persists for the remainder of the individual's life. I have watched, during many years, patients in whom it was present, and I have seen it persist in the same patient during sixteen years. In some cases it may appear, last for a few hours, disappear, and never recur; in others it may recur at frequent intervals for some weeks or months and then disappear. Many cases of paroxysmal tachycardia owe the paroxysms to auricular fibrillation, and in such cases auricular

fibrillation may last for a few seconds, a day or two, a few weeks, or months. As a rule, however, when the condition is intermittent in its appearance, the tendency to recurrence becomes greater, till finally it becomes permanently established.

In the great majority of cases of heart failure with dropsy the onset of heart failure is due to auricular fibrillation.

Before setting out in detail the features characteristic of auricular fibrillation I should like to indicate briefly the kind of case in which the condition occurs. Let me say at once that 60 or 70 per cent. of all cases of serious heart failure with dropsy met with in practice owe the failure directly to this condition, or have the failure aggravated by its presence. The most common evidence of the condition is the presence of irregular action of the heart of a very disorderly kind. It is that form of irregularity so frequently met with in the elderly, and in patients whose hearts have been damaged by previous rheumatic infection. In the latter class the association of irregular heart action with mitral stenosis has long been recognised, and, on account of this association, the irregular pulse is sometimes described as the "mitral pulse," with which all clinicians are familiar.

While senile and rheumatic hearts are most frequently affected by auricular fibrillation, there are numerous cases in which it occurs in patients with no history of rheumatism, and of an age at which senile changes are not usually present in a marked degree. Other cases are found which have been labelled "delirium cordis." Digitalis may produce auricular fibrillation in those predisposed to it; violent exertion also may induce it; and many of the recorded instances of heart overstrain afford excellent examples of this state and of the heart failure which accompanies it.

The patient is frequently conscious of the onset of auricular fibrillation, and the sensation is usually characteristic.

Many people become conscious of the heart's action when it departs from its normal rhythm. Thus, extra-systoles are sometimes recognised by the patient, who becomes conscious of the long pause which follows the extra-systole, or of the big beat which often comes after the pause. Patients liable to paroxysmal tachycardia recognise their attacks by the feeling of a gentle fluttering sensation in the chest. When the attacks of tachycardia are due to auricular fibrillation this fluttering is also present, but usually it is not a continuous fluttering, but is interrupted by thumping sensations, due to the occasional occurrence of bigger beats. When closely questioned these patients will generally state that the heart is irregular in its action.

The rhythm of the heart in auricular fibrillation is of a disorderly kind.

The symptom by which the clinical observer can most readily recognise auricular fibrillation is the character of the pulse. The pulse rhythm is usually irregular, and the irregularity is of a very disorderly kind. Irregularities, other than those due to auricular fibrillation, have usually a distinctive character, as, for example, the irregularity in the heart of the young, in which variations in rate coincide with phases of respiration, or the intermittent pulse, due to extra-systoles, in which the irregularity breaks in on an otherwise regular rhythm. In auricular fibrillation the pauses between the beats are, as a rule, continually changing, and two succeeding beats are rarely of the same strength, or the pauses between two succeeding beats of the same duration. The character of these irregularities will be made

clear by the radial tracings in Fig. 16. These tracings were taken from five patients. It will be found that the duration of the beats is always varying and that the rhythm is quite disorderly.

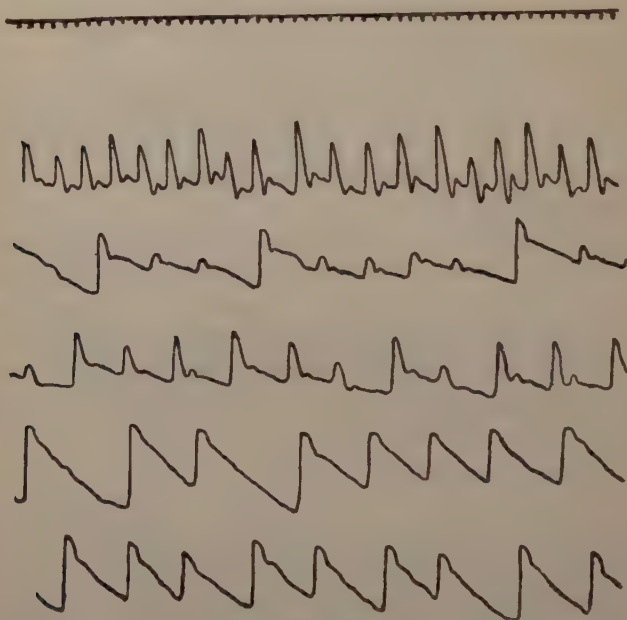


FIG. 16.

Tracings of the radial pulse from five different patients, showing the disorderly irregularity characteristic of auricular fibrillation.

When there are murmurs due to mitral or tricuspid stenosis, the presystolic murmurs due to auricular systole disappear with the onset of auricular fibrillation, while the diastolic remain.

I have already said that, with the onset of auricular fibrillation, all evidence of auricular systole disappears. When the auricles fibrillate in a patient with mitral stenosis, whose mitral stenosis has been revealed by the presence of a presystolic murmur alone, or by a presystolic accompanied by a diastolic murmur, the presystolic murmur disappears, but the diastolic remains,

as already described. When the heart is beating rapidly the diastolic murmur may fill up the interval between the second and first sounds, and many are apt to assume on this account that the murmur is presystolic in origin. But if the heart be auscultated after its rate has decreased—for example, under the influence of digitalis—the murmur then will be found to follow the second sound, and there will be an appreciable silence before the first sound at the exact moment at which the presystolic murmur was wont to be heard (see Figs. 7 and 8).

The onset of auricular fibrillation is usually accompanied by signs of heart failure.

While these are the signs by which we may detect the presence of auricular fibrillation, it is necessary to realise that it is the signs of heart failure itself which usually first attract attention. These signs of heart failure, however, are not peculiar to, or characteristic of, auricular fibrillation, but are common to heart failure induced by other conditions. They may range from a slight breathlessness on exertion to dyspnoea of the most severe kind, accompanied by dropsy and enlarged liver.

As a rule, the onset of symptoms of heart-failure is slow and gradual. This is due, in a measure, to the fact that the individual afflicted usually persists in living in his ordinary manner, though his heart is hampered by the abnormal action. On the other hand, the onset of heart failure may be very rapid. Within a few hours of the occurrence of auricular fibrillation the distress of the patient may be very severe, his countenance dusky, his heart dilated; he may also suffer from orthopnoea and a feeling of distress. I have seen these phenomena arise rapidly in cases where the fibrillation occurred intermittently, and the relief experienced by the patient when the heart resumed the normal rhythm was as remarkable and striking as was the onset

of the suffering. At once the patient knows that the heart's action has altered; he breathes in an easier manner and the feeling of distress disappears. Within a few hours the heart and liver have become reduced in size, the lividity of the face has passed away, and the tenderness in the chest wall and over the region of the liver has begun to disappear.

In many cases the onset of heart failure is accompanied by wasting, the patient sometimes losing a good deal of weight in a few months. There is usually, also, a tendency to flushing of the cheeks; the flushes are of a dusky colour, and occasionally a slight sallow tinge of jaundice may appear.

The prognosis in auricular fibrillation depends on the condition of the heart muscle and its power to maintain an efficient circulation.

We must bear in mind that auricular fibrillation is, in reality, the result of some process in the heart the precise nature of which is not yet fully understood.

There is, however, good ground for supposing that an essential part of this process is the escape of the auricle from the control of the sino-auricular node, the function of which is to secure a rhythmic and efficient contraction of the auricle as a whole.

In discussing auricular fibrillation as a clinical entity one is really putting forward the manifestation, or result of this process as if it were in itself a disease.

From the clinical point of view this has certain advantages, for the occurrence of auricular fibrillation induces such a profound change in the heart's action, and so powerfully affects its efficiency, by reacting on the ventricle and modifying the behaviour of that chamber to drugs, that we are compelled to look upon the condition as a state apart. Considering the variety of circumstances that induce auricular fibrillation, it is difficult to state briefly its prognostic significance. In

referring to the pathological lesions associated with it I showed that they were of a very diversified kind in nature and in degree. It is, in all probability, the extent of those pathological changes which determines the prognosis of auricular fibrillation, and an attempt should be made to estimate their extent. If we look upon the inception of a new rhythm as, in itself, embarrassing the heart in its work, then the maintenance of an efficient circulation depends on whether or not the heart is able to do its work when hampered by the new rhythm. The efficiency of the heart muscle is the fundamental matter. The question as to how efficient the heart muscle is must therefore be answered before any other question. Upon this answer everything really depends. We realise this, I think, very clearly when we consider the effect of fibrillation upon many of its victims, with what extraordinary rapidity heart failure sets in, how the patient becomes breathless and has to sit up in bed, how his face becomes livid, his heart dilated, and his liver swollen, and all this within a few hours of the onset of fibrillation. With the restoration of the normal rhythm these symptoms, as has already been said, quickly disappear. When fibrillation becomes permanent in such individuals the signs of heart failure persist sometimes in spite of all treatment, till death supervenes in a few weeks or a few months.

On the other hand, I have repeatedly seen fibrillation set in without the individual being in any way conscious of its presence. These cases may go on for years with little inconvenience; the majority, however, after some years, gradually show signs of a limitation of the field of cardiac response, and their future then depends on how they respond to treatment and on their ability to diminish their amount of bodily work and to live within the limits of the heart's strength.

Much more frequently there is a considerable limitation of the heart's power of response to effort, and if the usual life of the individual be pursued, without

appropriate treatment, there is a great tendency for the heart to fail gradually.

There is no doubt, also, that the onset of fibrillation can lead directly to a fatal termination, or, rather, that it can be associated with conditions which lead to death. Thus, one of my patients died suddenly forty-eight hours after the inception of auricular fibrillation. Another one fell down dead six months after its inception. I have seen a number of other patients, who had auricular fibrillation, die suddenly, but some of these suffered from a considerable degree of heart failure. It has appeared to me probable that in these cases the ventricle passed into fibrillation, as MacWilliam has suggested.

The usual approach to death in auricular fibrillation is by way of a steady advance of the heart failure, as shown by the breathlessness on exertion, orthopnœa, dropsy, and enlargement of the liver, etc., sometimes with an absolute failure of response to all forms of treatment. I have seen death ensue in this manner a few weeks after the inception of fibrillation; other cases have drifted on for a few months, while some have led a somewhat chequered career for a number of years, during which they were seldom fit for much bodily exercise.

In all cases of auricular fibrillation the possibility of the occurrence of embolism must be taken into account. Clotting of blood is liable to occur in the auricular appendices during fibrillation and thrombi may easily become detached.

This is peculiarly apt to occur on the resumption of the normal rhythm after a spell of fibrillation, the first full contraction of the auricle throwing out numerous fragments of blood clot into the general circulation.

In giving a prognosis in cases of auricular fibrillation it is therefore necessary to appreciate a good many factors over and above the mere presence of the fibrillation. It is necessary to form an opinion regarding the

extent of the changes that have led up to the fibrillation, and, in many cases, to find out how long these changes have been going on. This survey would include a knowledge of the date of any attack of rheumatic fever the patient may have had, and also how he comported himself before the onset of fibrillation, and whether or not he was liable to attacks of heart failure. These attacks of heart failure are important, because they point to a tendency to exhaustion which may be aggravated by the fibrillation. These questions are answered by a study of the functional efficiency of the heart and by the results of treatment. If aortic regurgitation is present with heart failure the outlook is not good, as such cases usually drift, and respond but slightly to treatment. The character of the murmurs present in mitral stenosis will also, as stated above, shed light upon the condition.

It is necessary, also, when auricular fibrillation sets in, to observe the accompanying changes in the heart and the manner in which it maintains the circulation. Thus, an increase in the size of the heart, or a rate over 120 beats per minute, usually leads to a speedy exhaustion of the myocardial strength. I have occasionally met with a patient having a heart-rate of 100 and 120 per minute, yet having no increase in the size of the heart and suffering little inconvenience; as a rule, however, any rate over 90 beats per minute tends to induce dilatation and consequent exhaustion. When, on the other hand, there is little increase in the rate, or when the rate is somewhat slower than normal and the response to effort good, the prognosis is usually very favourable.

A most valuable aid to prognosis may be found in observation of the manner in which the patient responds to treatment. In sudden attacks of severe heart failure, when the heart's rate is over 120 per minute, it will be well to suspend judgment until the reaction to digitalis is observed. Many such cases respond speedily

to digitalis, and, with the resultant decrease in the heart's rate, a remarkable degree of recovery may ensue, so that the patient may be able to undertake laborious work so long as his heart's rate is kept down by digitalis. This would seem to imply that the exhaustion is mainly brought about by over-stimulation of the ventricle, and that the slowing, by enabling the ventricle to get more rest, enables it to regain a measure of strength. From this result we can also gather that the ventricular muscle must be fairly healthy, and we can estimate, within certain limits, the amount of healthy muscle by the degree of recovery. (For treatment, see Chapters XX and XXI.)

CHAPTER XIV

AURICULAR FLUTTER

A CONDITION allied to auricular fibrillation, and sometimes alternating with it, is that when the auricular contractions are extremely rapid, sometimes exceeding 300 per minute. These contractions are not normal contraction but seem, like fibrillation, to occur as the result of some process which is not yet sufficiently understood. Although the auricles beat at the rapid rate, it is only on rare occasions that the ventricles respond to every auricular beat. In most cases the ventricle responds to every second auricular beat, so that the pulse-rate is one-half the auricular rate, the rhythm being quite regular. In rare cases the ventricle responds to every fourth auricular beat, and the rhythm is still regular. In a good many cases the response of the ventricle to the auricular beats is continually varying, with the result that the radial pulse is irregular, in some cases rapid and irregular (Fig. 18), in other cases slow and irregular (Fig. 17).

The morbid conditions provoking auricular flutter are unknown. It occurs in some who have had rheumatic fever, sometimes during convalescence, usually not till many years after. It occurs in the final stage of heart failure in cases with long-standing disease of the heart—myocardial and valvular. It occurs in acute infections of the heart, and in those with senile changes, and not infrequently it occurs in people in whom we can detect no history indicating a provoking cause.

The course auricular flutter pursues is variable. It may be present intermittently, the attack lasting a few seconds, a few minutes, a few hours, a few days, a few

weeks, or even a few months. Under these circumstances it is recognised as paroxysmal tachycardia (see pp. 136 *et seq.*). In others it is persistent and may go on for years.

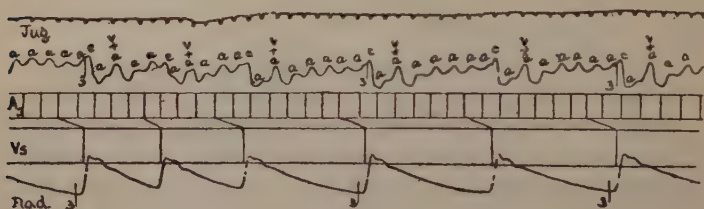


FIG. 17.

Auricular flutter. The upper tracing from the neck shows a series of waves, *a*, in the jugular vein at the rate of 220 per minute. The waves *c* are due to the carotid pulse and correspond with the radial pulse-beats (lower tracing). The radial pulse is slow and irregular, 35-40 beats per minute. In the intercalated diagram the downstrokes in the space *A*, represent the auricular beats, and the downstrokes in the space *V*, the ventricular beats which correspond to the radial pulse, and it shows that only a few of the auricular beats get through to the ventricle at irregular intervals.

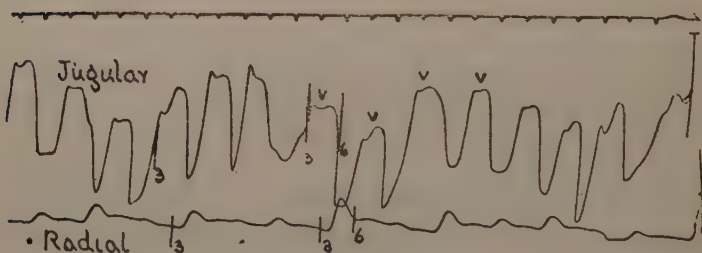


FIG. 18.

Tracings of the jugular and radial pulses with auricular flutter. The radial pulse is irregular, and the jugular pulse is of the ventricular type and gives no indication of auricular activity; see Fig. 19.

The effect on the heart's efficiency is also extremely variable. Some are not conscious of any limitations, while all degrees of inefficiency up to extreme heart failure may speedily arise after the occurrence of flutter. The degree of inefficiency depends chiefly on two things,

the rapidity of the ventricular contraction, and the state of the ventricular muscle. With great rapidity of the heart's action the output is so slight that the brain is insufficiently supplied with blood, and unconsciousness may result (Fig. 5), or the heart may dilate, followed by all the objective signs of heart failure. If the ventricles are diseased they may be unable to cope with the embarrassment of this abnormal rhythm, and grave failure of the heart may speedily arise.

It is often impossible to be certain of the nature of the condition by any method of examination, except by the

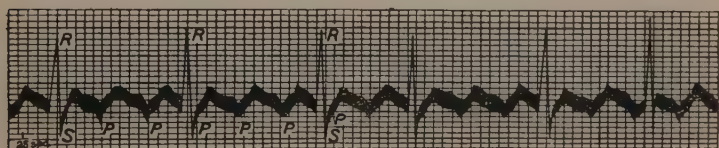


FIG. 19.

Electro-cardiogram taken from the same patient as in Fig. 18. The auricular movements (*P*) are regular and 310 per minute. The ventricular movements (*R*) are 120 and irregular.

electrocardiograph. It is chiefly to this instrument that we owe its recognition (Fig. 19).

It will thus be seen that the signs by which we recognise auricular flutter, the circumstances under which it arises, and the effects upon the heart's efficiency are extremely variable, so that, altogether, in auricular flutter, we have to do with one of the most elusive conditions in cardiac symptomatology.

Symptoms.

We can sometimes detect the condition by the rapid beating of the jugular veins in the neck (Fig. 17), but in many cases there is no jugular pulse, or it is of a kind that does not show these waves, as in Fig. 18. This is due to the fact that the rapid auricular contractions

vary in force, sometimes sending distinct waves in the jugular veins, sometimes not doing so, and then the waves in the jugular may be due to the ventricular systole.

A persistent rapid radial pulse of 150 or over, when the patient is at rest, and not increased by effort, usually indicates the presence of auricular flutter. This absence of response to effort is also typical when the rate is 120 or over, though effort in these cases may produce a sudden doubling of the pulse-rate. A rapid pulse-rate over 120 with the *normal rhythm* of the heart invariably varies with rest and effort.

Prognosis.

From the description given of auricular flutter it will be seen that it may impair the functional efficiency of the heart, but is not in itself a dangerous condition. When danger arises, it is in a heart which is already the seat of somewhat extensive disease, especially when affecting the ventricle, or in valvular disease with impaired ventricles. Hence, the already weakened heart is unable to cope with the burden thrown upon it by the abnormal rhythm. On the resumption of the normal rhythm there is also the danger of embolism, which was referred to in the case of auricular fibrillation. In giving a prognosis, each case must be carefully studied by itself, and the condition of the ventricle and the valve defects estimated. The manner in which the efficiency of the circulation is maintained, before and after the inception of the abnormal rhythm, and the response to digitalis must be particularly considered. There is danger when attacks of loss of consciousness arise, due to the rapid and inefficient action of the ventricle, with grave signs of heart failure. One patient whom I saw had an attack with congestion of the lungs, and died 24 hours later during a second attack. In many cases the flutter passes into permanent auricular fibrillation.

Treatment.

The treatment of the transient attacks is discussed under the heading of paroxysmal tachycardia.

When the condition is permanently established or persists for a long time, a course of digitalis should be tried, the digitalis to be given in the manner described in Chapter XXI. Digitalis is often beneficial and may cause the auricles to revert to the normal rhythm. In other cases it may only slow the heart. The slowing of the heart may be due to an increase of the heart-block, and with the stoppage of the digitalis the rate may gradually increase. If it is possible, the dose which will keep the ventricular contractions at a moderate rate (70 to 90 beats per minute) should be found, and the drug given as required. If this line of treatment be pursued, not only may it keep the heart in good condition, but it may lead to auricular fibrillation, and the restoration of a normal rhythm (Figs. 22-24); while if there be extreme heart failure, it may restore a more efficient circulation.

When auricular flutter is present and there is a tendency for the ventricle to follow each auricular contraction, the quieter the patient can be kept the better, and sedatives such as the bromides should be given to ensure sleep and to prevent mental disturbances, dreams, excitement, etc. As in auricular fibrillation, we know of no remedy which will with certainty stop this abnormal rhythm in all cases.

CHAPTER XV

HEART ATTACKS

Palpitation, paroxysmal tachycardia, sudden failure of the heart overstrain of the heart.

A GREAT many people complain of "heart attacks." Since the attack may not be in progress when we see these people it is always necessary to ask them to explain what they mean by the expression. Some will describe as heart attacks conditions in which they feel faint or actually do faint. We can estimate the significance of such conditions by means of what has already been said on exhaustion and syncope. Others again complain of heart attacks to which they give the name "palpitation." As employed by patients, this term will be found to include many different phenomena. Thus, if extra-systoles occur rather frequently for a short period, the patient is quite likely to say he has palpitation. Others will describe how their hearts beat at a moderate rate but very forcibly, the force gradually subsiding. In others the heart beats rapidly, the rhythm being regular or irregular. If these patients are asked to indicate the nature of the action of the heart, for example, by tapping with the finger, they will often give a fair indication of the nature of their troubles.

The sensations peculiar to extra-systoles have already been described. Conditions in which the heart is increased in rate may be divided into two great groups—one in which the rhythm of the heart is normal, and the other in which it is abnormal. In order to differentiate between them I will call the first of these "palpitation," and the second "paroxysmal tachycardia." The distinction between these two (over and above the nature

of the rhythm, which cannot always be determined without instrumental aids) is that while in palpitation the onset is often gradual, but occasionally sudden, and the offset always gradual (the heart imperceptibly falling to the normal—sometimes quickly—sometimes slowly), in paroxysmal tachycardia both onset and offset are always sudden. In palpitation the slowing heart at offset can be increased in rate by exertion; paroxysmal tachycardia, on the other hand, terminates with one or more strong beats with prolonged intervals between them, and effort seldom has any effect on the rate, though on rare occasions it may double it.

Palpitation occurs with a normal rhythm of the heart, and is not generally cardiac in origin.

Palpitation may occur in relation to a great variety of complaints. The patient is usually conscious of the change in the heart's action; he feels the rapid beats and sometimes describes them as "gentle," sometimes as "hard and hammering." This latter sensation may occur with little or no increase in frequency. In cases of valvular disease, where there is a limitation of reserve force, slight physical effort or mental excitement may readily induce an attack. Even in the healthy, certain mental states may have the same effect. When the system is weakened from disease the liability to attack is naturally much increased. It is in certain neurotic subjects, particularly females, that one sees the complaint attain its most distinctive features. There may be no organic affection of the heart; and, though frequent attacks may ultimately induce exhaustion of the reserve force, yet, as a rule, they do not appreciably shorten life. Anything that startles the patient, whether it be a sudden noise, or mental perturbation, or uncomfortable dreams, readily induces an attack. But an attack may supervene as the result of more obscure causes, amongst which may be included reflexes from organs more or less remotely situated (stomach, uterus).

The sources of the trouble may, in other cases, be indiscernible. When a severe attack comes on, the patients may become painfully aware of the violent action of their heart. They prefer to sit upright; they draw deep inspirations and move uneasily from side to side, with the hand pressed over the heart. The attack is accompanied by sensations of a distressing nature, such as a sense of suffocation and a fear of impending dissolution. When it subsides it leaves the patient exhausted.

During the attack the pulse is usually increased in frequency. The artery may be of fair size; sometimes, however, it is very small. The impact of the pulse-wave on the finger is sudden and sharp, and of extremely brief duration.

We occasionally meet with patients in whom the pulse is extremely rapid for a period, sometimes for a few minutes, sometimes for a few hours. No sensation other than exhaustion is experienced during the attack, which quietly subsides. The causes of these attacks are so obscure that it would be mere guess-work, in the majority of cases, to discuss them.

The opinion given about the case should be based, in every instance, on careful consideration of the cause of the disturbance of the heart; an inquiry must therefore be made into the patient's state of health and into the condition of other organs, particularly the digestive organs. The treatment is directed to the removal of any exciting cause or to the improvement of the patient's health as a whole. Where the patient is of a nervous temperament the judicious use of the bromides will help in diminishing the frequency and severity of the attacks, especially of those attacks which occur in the night. During the attacks relief is often obtained by drinking a cup of strong hot coffee or tea.

Paroxysmal tachycardia is due to the sudden inception by the heart of a new rhythm.

As we get a clearer insight into the mechanism of the heart's action we are able to identify more clearly the

exact nature of cases which we were wont, in the past, to include under some general term. Paroxysmal tachycardia is a term used here to indicate the starting of the heart's contraction, at an abnormal place, with coincidentally a sudden increase in the rate of the heart, and finally a sudden cessation of the rapidity on resumption of the normal rhythm. Analysis of a great many cases shows that the cause of the attacks, in most instances, is a transient fibrillation of the auricle when the rhythm is irregular. In other cases they are due to auricular contractions arising at an abnormal source and occurring at a great rate (from 150 to 300 per minute) when the rhythm is regular ("auricular flutter"). In rare cases the new rhythm may start in the ventricle.

Symptoms.—The heart suddenly takes on a rapid action; this may last for a few beats or it may go on for minutes, hours, days, or weeks. When the attack stops it does so suddenly, not gradually, as in palpitation. The rate is usually increased, sometimes greatly. A patient may have but one attack, or his attacks may come on at frequent intervals in the course of ten or twenty years; again, they may be of great frequency and occur every few weeks or days; or there may be several attacks in one day. After one or two attacks the heart may settle down permanently to a fibrillation of the auricle or to auricular flutter.

The sensation felt by the patient when the rhythm is first started may be so slight as to pass unnoticed by him. Usually he is conscious of a curious fluttering sensation inside the left chest. This sensation is very characteristic and almost pathognomonic. It may be described in various ways according to the vocabulary of the sufferer, but the essential feature is the soft and gentle movements, which are not rhythmical, but which vary in intensity. This is in striking contrast to the sensations that may arise from stimulation of the heart during a normal rhythm—for example, in palpitation. Frequently the sensation is so disquieting that the patient rests, or walks about cautiously and quietly.

While the attack is in progress there may be but slight limitation in the field of cardiac response. In other cases the patient finds that the exertion he was wont to undertake with comfort now induces breathlessness. In rare instances the sense of prostration and of weakness is extreme and is accompanied by a profound fear of dissolution; sometimes loss of consciousness for hours may occur. In these instances the heart beats at a very rapid rate—up to 300 per minute—and the pulse is scarcely perceptible. Again, there are cases in which the associated heart failure may be so extreme that in a few days, or even a few hours, evidences of imminent peril are shown. The patient has to keep in bed; the dyspnoea is so great that he cannot lie on his back, but must be propped up. If he survives, oedema of the legs quickly supervenes, the lips become livid, and the face swollen. The pulse is small, rapid, and sometimes irregular, the beats varying remarkably in strength. The veins of the neck are often full and pulsate with great rapidity. The heart dilates in a few hours, sometimes extending two inches in the transverse direction. The sounds alter, becoming short and sharp, and if the heart's action is rapid, often no murmur can be detected. The liver becomes enlarged and may be found pulsating two or three inches below the ribs. The tissues covering the heart and the liver often become extremely tender on pressure.

With the sudden reversion of the rhythm to the normal the change in the patient's condition is even more remarkable than that attending the rapid onset of the symptoms of heart failure. At once the patient heaves a great sigh of relief, and in a very short time, within half an hour, all abnormal signs in the lips, face, and enlarged liver disappear, while in a few hours the heart may be beating within its normal limits.

In some the paroxysmal tachycardia may be accompanied by no marked change in the size of the heart. In these cases the patient is generally conscious of the

heart's abnormal action, and instinctively avoids active exertion, either by keeping in bed or by resting in a chair, or by walking about very quietly. The attack usually lasts for a few hours, but may occasionally last for many hours, or even one or two days. At the end of this time there may be no increase in the size of the heart and no signs of dropsy or enlarged liver.

Attacks of paroxysmal tachycardia may occur at all ages; but they occur more frequently in the later years of life. It is impossible to tell what predisposes to these attacks; a good number of patients give a history of rheumatic fever. The attacks may occur during convalescence from the fever or many years afterwards; senile changes in the heart also predispose to attacks. In a few cases disease in other parts of the body seems to provoke attacks.

Prognosis.—The prognosis in these cases is fraught with considerable difficulty. The symptoms during an attack may be so alarming that the inexperienced are apt to look upon a patient as hopelessly stricken. I remember being called one night to see a woman eighty years of age. I found her heart extremely rapid and irregular, her face swollen and livid, and she was gasping for breath. I told her friends the end was approaching; but, on calling to see her next morning, I found her walking out in the street. After that she had several attacks, and finally died during one of them. I have watched other patients for years, when the symptoms have not been so extreme and have given rise merely to a slight uneasiness in the chest. In these the heart has not dilated, though the rate may on occasion have reached nearly 200 per minute. Again, I have watched patients for over twenty years who had only one attack, and can testify that they have had no other.

In some the change, from being transient, becomes permanent; and herein lies the danger. For if the extreme form of heart failure which arises persists, in spite of treatment, the patient drifts to death.

The prognosis depends also on the degree of dilatation present. If the heart does not increase in size and the attacks are transient, then, on the whole, the prognosis is good, though the patient's life may be greatly crippled by continually recurring attacks. When, on the other hand, there is dilatation, with the accompanying symptoms of dropsy and enlarged liver, the outlook is bad.

Another element in the prognosis is the associated disease. In many cases there is a history of rheumatic fever, and valve lesions, the result of the fever, may hamper the heart. If mitral stenosis or aortic regurgitation be present, the extent of the damage and the degree of impairment must be considered. The frequent occurrence of attacks of paroxysmal tachycardia adds to the gravity of the condition. Senile changes in the heart may be associated with it; the extent of these must be gauged by the manner in which the heart responds to effort. When the response is good and the heart suffers little during the attack the prognosis is quite favourable.

Treatment.—Absolute quiet is essential during the attack. This suggestion is, indeed, scarcely necessary, for the patient usually seeks rest or moves about very quietly. In some cases, as I have said, quiet movement may occasion little distress. A great many recommendations as to means of arresting these attacks may be found scattered through medical literature. Because the attacks are commonly transient in duration, some remedy, employed opportunely, seems to the inexperienced onlooker to restore the heart to its normal action. Many patients themselves have a knack of doing something which seems to change the rhythm. Sometimes the attacks can be stopped by the patient by simply taking a series of deep breaths, by slapping the chest, by the sudden application of cold water, or by vomiting.

Drugs of the most diverse character have been described as active agents in stopping attacks; in this

category are nitroglycerin and adrenalin. In my early days I, too, thought I knew how to stop attacks; but more extended experience has shown me that, when they stopped, it was from some cause unknown to me, which was independent of any means I employed. I have tried intravenous injections of strophanthin, with no satisfactory result.

Many people who suffer from these attacks notice that, when there is digestive discomfort, the attacks are more readily provoked; undoubtedly the clearing out of the bowels and careful dieting diminish the frequency. In one case of chronic appendicitis the attacks disappeared after the removal of the appendix. Sometimes the bromides, pushed till apathy is produced, diminish the frequency of the attacks.

When signs of heart failure appear and the abnormal rhythm becomes permanent and is found to be of the nature of auricular fibrillation, or flutter, then the treatment described in connection with these conditions should be employed.

Sudden failure of the heart.

On rare occasions an individual with some organic lesion but otherwise in fair health is suddenly seized with intense breathlessness and may die in the course of a few hours. This phenomenon is seen as an end picture in some cases of aortic regurgitation. We also see the same fatal state in cases of acute oedema of the lungs, where frothy, blood-stained expectoration wells up out of the lungs. In some of these cases mitral stenosis is present; in others there is no valvular lesion. I am unable to account for the attacks.

The most frequent cause of sudden attacks of heart failure is the inception by the heart of a new rhythm. In auricular flutter and fibrillation the rate of the ventricle may be so rapid and the output may become so reduced that an insufficient quantity of blood is sent

to the brain. From this cause syncope results (page 25). In other cases there is no loss of consciousness, but there is much breathlessness, with great accumulation of blood in the venous system, as shown by lividity of the face, enlargement of the liver, and fullness of the jugular veins. Congestion of the lungs may be so extreme that in a few hours crepitations appear at the bases, with dullness to percussion. Such attacks are liable to be mistaken for pneumonia.

Overstrain of the heart.

There is a belief, widely cherished by the profession, that overstrain of the healthy heart is a frequent occurrence. Some years ago quite a scare occurred among doctors about the danger of overstrain from athletics and school games. For a number of years I had brought to me great numbers of boys, girls, and young men with "strained" hearts. In nearly every case the symptoms on which this expression of opinion was based could be referred to one or more of these signs (systolic murmurs, youthful type of irregularity, physiological dilatation of the heart) which I have already described and characterised as compatible with perfect health.

In a number of cases when grievous heart failure has suddenly set in after a severe effort, it will be found that the onset of heart failure was due to fibrillation of the auricle. The literature of the subject, when cases are described with sufficient clearness, reveals the fact that nearly every one of these patients had auricular fibrillation. Most of them are elderly people, or people with some old rheumatic history. I have, however, seen auricular fibrillation occur in a few young people after effort, in whose hearts I could detect no other fault.

I have described on page 152 the case of a youth in training who collapsed suddenly, and have pointed out, in connection with that case, that the trouble arose, not alone nor even chiefly from exertion, but from the

presence of some infection. During war the severe tests of endurance, which are a commonplace of campaigning, are said to provoke heart overstrain. An examination of a large number of cases shows that an infection or other predisposing influence—for example, want of sleep and worry—combines with the overstrain to produce the exhaustion, and that in no case are the symptoms purely cardiac in origin (page 151). When, therefore, we encounter exhaustion in a hitherto healthy individual, it is our duty always to search for some cause other than mere overstrain.

I do not say that exertion alone may not damage the heart, but I do assert that such an event is extremely rare. In a few cases, in which I could find no provocative cause other than prolonged violent effort, the chief complaint was pain or distress in the chest; there was no abnormal action of the heart and no increase in its size.

CHAPTER XVI

ACUTE AFFECTIONS OF THE HEART

There is a great similarity between the symptoms of an acute affection of the heart and those due to fever and the toxins of organisms in other parts of the body.

It is well known that, when the temperature is raised, certain alterations take place in the circulatory system. The rate of the heart, for instance, increases and the peripheral vessels dilate. These phenomena may arise simply from the effect of heat; and, indeed, we get the same results from a hot bath. It is often assumed that the temperature alone is the cause, and attempts have been made to associate a definite increase in heart-rate with each degree of raised temperature. This view, however, is fallacious, for while, no doubt, increase of the temperature does modify the heart's rate, the influence of the agent causing the rise of temperature has other effects. I have kept notes of individual patients, and I have found that, during febrile attacks of different kinds, the rate of the pulse did not correspond with the degree of fever, but varied, seemingly, with the nature of the infection causing the fever. An attack of ague furnishes striking support of this view. In ague, during the phases of the hot and cold stages, marked variations are found in the heart and peripheral vessels, yet the rise in temperature is constant. Again, in typhoid fever the pulse-rate may actually fall while the fever is maintained.

The combined reaction of temperature and infection on the heart and blood-vessels is extremely varied. In many cases, beyond increase in cardiac rate, no change

can be detected; while, in other cases, marked changes are found in the heart—for example, dilatation, sometimes to a considerable extent, and the appearance of systolic murmurs at the apex and the base.

These phenomena may only be the physiological response of the heart to certain stimuli, such as the toxins of disease organisms in other parts of the body. On the other hand, these signs may be evidences of the invasion of the heart by the disease agents, and the production by these agents of destructive lesions of the tissues of the heart. It is often a matter of difficulty to decide the nature of these phenomena during the fever, and it may be necessary, in many cases, to suspend judgment till the cause of the illness has been ascertained and more clear evidence gained. I mention the matter in order to direct attention to the fact that these phenomena may arise in cases in which serious mischief has not occurred; this should be borne in mind in every case. It not infrequently happens that their appearance in the course of a febrile attack, such as rheumatic fever, is taken as an indubitable sign of endocarditis, when, as a matter of fact, it has no such significance. I repeat that it may not be possible to decide the matter until after the subsidence of the fever, when the heart's rate has fallen and its size diminished; in the young the youthful type of irregularity may appear during convalescence, and by its appearance testify to the escape of the heart. On the other hand, a persistent increase in the rate of the heart may occur, pointing to the presence of some active mischief in the organ.

From such considerations we gather that there are two conditions of the heart which may supervene upon infection: (1) That in which the heart itself is invaded by some organism whose presence produces certain signs and causes actual damage to the cardiac tissues; (2) That in which an organism present in other organs and tissues affects the heart, not directly, but by the toxins it has produced. The distinction is of the very

highest importance, because the latter condition, which I shall call the "poisoned heart," is of frequent occurrence; its symptoms are often misunderstood.

The heart in rheumatic fever.

Acute affections of the heart are seen most frequently during an attack of rheumatic fever. In rheumatic fever the pain in the joints and the illness produced by the fever first call attention to the condition. I need not dwell on these phenomena, as they are well known. The frequency of heart-disease in rheumatic fever leads us to study the heart carefully, even in the mildest forms of the complaint, among which must be classed the "growing pains" of the young. The symptoms of the invasion of the heart are varied. There is often an increase in size; sometimes this increase is slight, sometimes it is of very great extent. This latter state of affairs is found particularly when the myocardium is invaded. Invasion of the myocardium is, of course, of frequent occurrence; nevertheless, there is nothing to distinguish the enlargement caused by it from the dilatation of the poisoned heart to which I have referred, except in those rare instances when an irregularity of the heart's action, due to partial heart-block, demonstrates that the auriculo-ventricular bundle has been invaded.

The appearance of a soft systolic murmur warns us that the endocardium may be invaded. With changes in the character of the murmur this warning becomes confirmed. The appearance of a diastolic murmur causes us to recognise that the aortic valves have been invaded and damaged. I may remark here that the presystolic murmur of mitral stenosis never can appear during the first attack, as it is the outcome of a slow cicatrising process which develops after the acute inflammation has subsided. Invasion of the pericardium is recognised by the peculiar pericardial friction sound,

or by the characteristic signs of effusion into the pericardial sac.

On rare occasions similar phenomena arise in connection with other acute diseases—for example, septic joint affections, influenza, erysipelas, pneumonia.

The treatment of acute affections of the heart.

As soon as we recognise that we have to deal with rheumatic fever we must, as a matter of routine, place the patient in the most favourable condition for recovery; we must also employ appropriate remedies for his relief and attempt so far as we can to stay the progress of the disease (*e.g.* by the use of the salicylates).

In all cases it is a good rule to keep the patient strictly in bed until there is assurance that the condition is quite abated. In spite of this measure, however, difficulty will often arise. The presence of a murmur and of irregularity of the heart is so often associated with rheumatic fever that, when these occur in the course of this condition, or of other febrile states, it often happens that they are taken as signs of disease. The signs are, in fact, perfectly innocent and have already been dealt with.

On the other hand, so long as there is positive evidence of mischief—for example, a persistent increase of rate, a changing murmur, or a diastolic murmur which has arisen during the illness—the patient should be kept at rest, and this should be maintained until the rate is quite normal, even although six months may elapse before that result is brought about.

During an attack of acute rheumatism and in the subsequent convalescent stage, treatment of the heart by drugs is not indicated, and the indiscriminate use of the so-called cardiac tonics (*digitalis*, etc.) is more likely to do harm than good.

Septic Endocarditis.

There are a great many septic infections which injure the heart, the specific organism invading the organ.

The endocardium is frequently attacked, and the disease is then described as septic endocarditis. In these cases there may be an invasion of the myocardium, resulting in extreme impairment of the heart muscle.

The illness in these cases usually begins insidiously, and at first may be mistaken for some trivial febrile complaint or for influenza. Soon, however, the extreme prostration of the patient, the recurrence of rigors, and the patient's own sensation of illness show that the condition is of a more serious nature. Usually, also, there is an excessive perspiration. If the heart be watched it will be found to dilate, and a systolic murmur appears. The true nature of the condition may not be revealed until the detachment of a vegetation produces hemiplegia, or an infarct in the spleen or kidney; death may speedily supervene (malignant endocarditis).

Other cases may linger on with indefinite febrile attacks; the patient is sick, pale, and ill, and the real nature of the illness remains a mystery. In some of these cases very little change takes place in the heart. I have seen a woman, after confinement, have slight fever for nine weeks without resulting change in the size of the heart; her heart-rate was generally about 80 per minute, and a rough systolic mitral murmur was the only abnormal sign present. An attack of hemiplegia led to the recognition that the rough mitral murmur was due to vegetations on the valves. Osler published an account of ten cases of chronic infective endocarditis. In addition to the irregular fever he gives the following as the most suggestive features which help to identify the nature of the disease: (1) A knowledge of the existence of an old valve trouble; (2) the occurrence of embolic features, sudden swelling

of the spleen, sudden attack of hæmaturia, embolism of the retinal arteries, hemiplegia, or the blocking of a vessel in one of the limbs; (3) the onset of special skin symptoms, purpura, and more particularly the painful erythematous nodules, in all probability due to minute emboli; (4) the progressive cardiac changes, the gradual increase in the dilatation of the heart, the marked change in the character of the mitral murmur, the onset of a loud, rasping, tricuspid murmur, or the development, under observation, of an aortic diastolic bruit.

✓ In pyæmia and puerperal septicæmia we encounter conditions of profound gravity which are caused by certain organisms. The pulse in these cases gives the most trustworthy information. It is small, soft, and easily compressed, not necessarily very rapid; the temperature need not be high (101.2° F.). The heart shows little change except that its sounds are feeble; the patient is lethargic, the face is slightly sallow or pale and sunken. The aspect of the patient, taken with the rate of the pulse, affords assistance in recognising the condition. I dwell upon this point because, happily, some of the younger members of the profession have little experience of dangerous forms of puerperal fever, but, having heard of the terrors surrounding them, are not infrequently unnecessarily alarmed at the occurrence of a post-partum rise of temperature of trivial significance; others, again, do not recognise the significance of the heart symptoms when danger actually arises.

The outlook in these cases is bad, and all attempts at treatment have hitherto proved of no avail.

The Poisoned Heart

The toxins produced by germs invading the body may affect the circulatory as well as other systems.

When an individual suffers from the invasion of his body by some microbic infection he falls into a state of ill-health. The symptoms are at first subjective and

give rise to a vague feeling of being "out of sorts." If the infection be severe and belong to some of the well-recognised groups of infection (*e.g.*, typhoid fever, pneumonia, scarlet fever, or measles) the symptoms characteristic of these diseases manifest themselves, and all the symptoms of ill-health are naturally attributed to the specific infection. But there are a great number of obscure illnesses which never develop symptoms that, in our present state of knowledge, we can recognise as belonging to a specific disease. These illnesses may be of a mild type and may give rise only to ill-defined sensations. Some of the sensations may be referable to the circulatory system—for example, palpitation, breathlessness, or exhaustion easily provoked; while abnormal signs—for example, increased rate, systolic murmurs, or dilatation—may appear in the heart. In the absence of absolutely definite signs of infection the patients are often regarded as the victims of an affection of the heart.

One feature which distinguishes these cases from cases suffering from primary cardiac failure is a feeling of tiredness always present and a sense of exhaustion easily induced. If these people be asked what is the sensation which they, on making a mild effort, first recognise as indicating distress, they will invariably reply, "a feeling of exhaustion." It may require a violent effort—for example, running up stairs—to induce palpitation or breathlessness, and therefore the onset of these symptoms is often attributed to such an effort. The sensation of exhaustion is, however, the predominant feeling; it comes on, too, at unexpected times, as well as during effort. (See Chapter VI.)

The peculiar condition described as the irritable heart of soldiers illustrates the effects of toxins with conditions producing exhaustion.

The importance of recognising the nature of the source of exhaustion and of differentiating between the

exhausted state and the state of cardiac inefficiency was brought prominently before me in an investigation which I, along with others, conducted on soldiers who had been invalided because of a heart affection. Since the American Civil War it has been understood that many soldiers break down in a peculiar way; they show no organic lesion, and as, in a considerable number, the heart is liable to periods of rapid action, their condition has been described as the "irritable heart" of soldiers. The well-written accounts of these cases show that they corresponded to the cases we met with in our investigation. In an analysis of 400 cases we found that 90 per cent. did not suffer from organic disease of the heart, and that, although increased rate, dilatation, and other abnormal signs were sometimes present, those signs were only part of a general condition. In nearly all we could trace the onset of illness to some infection (as shown by fever or diarrhœa). These men led strenuous lives while they were suffering from the infection; after a time, however, they got weaker, and finally they collapsed, and were diagnosed as "heart" cases. In every one of these cases the chief symptom was a feeling of weakness—"rotten" was an expression commonly used by the men to describe their sensations. I asked many of them what pulled them up when they were walking on the level, and each one said it was the sense of exhaustion; if they ran or went uphill they felt short of breath and suffered from palpitation.

It is manifest that this class of patient does not suffer primarily from a heart affection, but rather from some toxic condition affecting the central nervous as well as the circulatory system.

Case illustrating the effects of exertion during a mild febrile attack.

In order to make clear the nature of this sudden exhaustion from infection I cite the following experience.

A youth training for a race had been spending some time each day running round the track. One day while so engaged he felt ill and giddy; he stumbled and fell and was picked up partly unconscious. His doctor at once said that he was suffering from heart overstrain; and a slight dilatation of the organ confirmed him in this view. I was asked to see the young man. His account of himself was that he had been perfectly fit until the morning of his seizure. When he awoke on that morning he felt "rather seedy." Notwithstanding this, he started on his usual training exercise, with the result just described. Now, no sound heart gives out in this way from overwork, as will be readily gathered from the account I have given of what constitutes heart failure. When such sudden attacks of weakness occur in presumably healthy people we have to look for some other cause—in the elderly the onset of a new rhythm, such as auricular fibrillation; in the young some complaint other than heart failure. As in the case I have mentioned the training up to the day of the breakdown testified to the health and vigour of the patient's heart, I expected to discover an infection. I took the temperature and found it 100° F. It was thus evident that the weakness was the outcome of some slight febrile attack. After a few days' rest the patient was quite well, and shortly after he resumed his training. In the end he ran his race with no sign of weakness.

Such is the account of a typical instance. In cases where a sense of exhaustion is easily provoked, and is accompanied by palpitation and breathlessness on exertion, the physician should always inquire how long it is since the individual was in robust health, and particularly in what circumstances he first recognised his weakness. Frequently he will find that the onset of the ill-health dates from some illness, it may be of a trivial nature—such as influenza, a mild attack of diarrhœa, or some indefinite condition with a little fever.

A great variety of conditions may induce exhaustion and affect the circulatory system.

There are other patients from whom we cannot get a definite history as to the beginning of the trouble. In these cases we must exercise care to examine the condition of the other organs. It may be laid down that the intestines furnish the most common provoking cause. Many different morbid conditions of the gut are closely associated with the circulatory system—for example, dilatation of the stomach, with and without pyloric stenosis, stasis in some portion of the intestinal tract, constipation, and so forth. It is not, of course, the organic mischief present which directly causes the patient's complaint; but the organic change interferes with the healthy action of the digestive tube, and so, by retaining the contents of the tube, permits decomposition to be set up. The products of this decomposition are absorbed and act as the poisoning agent. Other sources of poisoning are found in chronic local inflammation—*e.g.*, a chronic appendicitis, or abscesses in different regions of the body. External agents also produce these effects upon the circulatory system, as may be seen in persons who over-indulge in alcohol and tobacco. It is scarcely necessary to insist that unhealthy occupation or surroundings, with insufficient food, are predisposing factors.

Symptoms.

The symptoms of the condition in the milder cases are not those of a poisoned heart alone, but are also those of disturbance of other systems. This applies especially to the nervous system. Thus, the most frequent symptom of all is absence of the sense of well-being: the patients are conscious that something is amiss, and many of them, as I have stated, express this feeling colloquially by the word "rotten." There is often an irritability of temper which is at times unaccountable; frequently, also, there is great mental

depression. Signs of vaso-motor irritability are commonly met with. The appetite is fickle and frequently poor; and nausea may be provoked. The response to effort is limited, the limitation being shown in the majority of cases by a sense of exhaustion. Breathlessness on exertion is usually present also, though it is not so much complained of as is exhaustion. The peculiarity about the limitation of the strength is that it is so very variable. In favourable circumstances, the individual at times can do a surprising amount of work, while at other times his strength is speedily exhausted. Also, the sense of exhaustion is quickly recovered from; when the patient sits or lies down it passes away in a few minutes, and he may later undertake much effort without suffering. In extreme cases the cardiac inefficiency may be very marked and may lead to great breathlessness and to extreme heart failure with dropsy and enlarged liver. Such extreme cases rarely arise as the result of microbic invasion, but are frequently met with in alcoholics, especially in cases where the alcohol consumed has been contaminated with arsenic. Pain is a very frequent symptom. It is referred to the chest, over and internal to the left breast. It is usually not severe, and is commonly rather a sense of dull aching than a pain. Occasionally, however, it may be of intense severity and may assume all the characters of a violent attack of angina pectoris. One feature, however, distinguishes it from the pain due to over-exhaustion of the heart as a result of actual disease, and that is its tendency to occur when the individual is at rest. So striking is this tendency that I have laid it down for many years that, when pain with the characteristic cardiac distribution occurs *only* while the patient is at rest, the graver conditions of disease may be excluded and search instituted for some provoking cause, especially for some source of poisoning. In many cases of poisoning the individuals suffer no pain, even when making a considerable effort. Effort, on the

contrary, may, in other cases, provoke the pain. In these latter cases the fact that the pain also comes on when no effort is being made suggests that there is some toxic influence at work. In rare cases of tobacco poisoning the pain is provoked during the act of smoking. This is seen typically in elderly men who are actually suffering from disease of the heart; occasionally, however, we find it also in the young who have no cardiac disease of any kind.

Physical signs.

The patient may present no physical signs. In the majority of patients, however, there are evidences of an unstable vaso-motor system—for example, a tendency to flush and grow pale. The least excitement in these cases causes the face to become red, while the hands and fingers remain pale and cold. Sometimes the pallor of one or more fingers is extreme, they become numb and “dead.”

The heart's rate may be persistently increased, or may be easily provoked to an increased rate; attacks of palpitation are also easily provoked. Mental excitement or effort are the usual stimulating factors, but the increased action of the heart may occur without the presence of any ascertainable cause. The heart's size may be unaffected; on the other hand, a slight increase in size is not infrequent. It is only in the extreme cases, which are rare, that we get dilatation of the heart; and then we may also get large pulsation in the veins of the neck, dropsy, and enlargement of the liver.

Murmurs are frequent; they are usually of indefinite origin, though some seem distinctly to arise at the mitral orifice. They are always systolic in time.

Prognosis.

The outlook in most cases is distinctly favourable. I have never seen a single case of the milder form of the condition develop any grave sign of heart failure.

Many gradually get rid of the poison and recover; on the other hand, some are crippled for years. The one danger resides in the fact that, being in a state of impaired health, these patients are less resistant to other infections, and so are apt to fall a prey to them. I have seen a few cases, in which the source of infection was originally intestinal, develop consumption.

Treatment of the poisoned heart.

It is scarcely necessary to dilate on the principles of treatment. The recognition of the condition compels a search for the source of the poisoning and the adoption of measures for its removal, whether it be a focus of infection, such as an abscess, intestinal absorption, or alcohol. In cases which are the result of a past infection, the placing of the patient under hygienic conditions best calculated to restore his general health is indicated, rather than attempts to treat the heart alone.

CHAPTER XVII

BLOOD-PRESSURE

Blood-pressure records are used for purposes they cannot fulfil.

IN discussing murmurs I pointed out that the un-reasoning way in which the profession took up the stethoscope had hindered the advance of medical science and had done grave injury to many individuals, although no doubt a good deal of useful knowledge had also been acquired. We can see the same influence at work in the attitude of the profession to almost every new method, notably at present in regard to blood-pressure instruments. Although it is only a few years since approximately accurate instruments have been in use, important and far-reaching conclusions are drawn which are not only quite unjustified, but are even misleading. For since we are ignorant of some essential factors concerned in the production of the blood-pressure we do not yet know what are the normal variations in health, let alone the variations that occur in ill-health. We find the sphygmomanometer used as a basis for diagnosis, as a guide in treatment, and as providing grounds for a prognosis, even in life insurance.

We find it used to estimate the efficiency of the heart muscle—sometimes by estimating the relation of the systolic to the diastolic pressure, sometimes by the variations in blood-pressure by change of posture or by effort. Such calculations as these are based on a misconception of what heart failure is and what part the heart muscle plays in the production of blood-pressure.

The research into the significance of blood-pressure has not been undertaken by the observers who have the appropriate opportunity.

It is possible that when we have sufficient knowledge of the blood-pressure we shall find it of considerable help in our work, but there is no prospect of our acquiring that knowledge in the near future. It has not yet dawned on the great majority of writers on this subject how this knowledge is to be acquired. While many of the factors concerned in its production may be discovered by investigation in the laboratory and the hospital ward, other equally essential data cannot be acquired in these places. I have endeavoured to watch the variations in blood-pressure, as estimated mechanically, in people for a great number of years, and have been struck by the great variations that may occur even in healthy individuals. The earliest complaints which caused some patients to consult me were accompanied by a high blood-pressure, and the progress of their ill-health has been rapid. In a few patients death has followed in a few years in spite of all kinds of treatment. Others with the same high blood-pressure have not gone so rapidly bad, but have lived for long years with a very gradual deterioration in health, while others have pursued a normal life with no signs more remarkable than would accompany advancing years.

I have no knowledge of the antecedents of these cases, and am unable to say what value should be attached to the blood-pressure records. Some slight advance I have made, in that I never take the blood-pressure alone as a guide to an opinion, but consider also the state of the arteries, the size and efficiency of the heart, and the state of the kidneys. With the data thus obtained I venture upon an opinion, but even then it is by no means a very certain one.

The study I have given to this subject leads me only so far as to indicate on what line the subject should

be pursued. The observers who alone are capable of undertaking this quest are young practitioners in general practice, because they have the two great essentials—the opportunity for watching individuals for many years and the prospect of living long enough to complete their observations. It is manifest that before we can recognise the significance of an abnormal state we must know the normal and the limits within which variations may occur in health. Many writers on blood-pressure are teachers of medicine, and if they would but train their students how to observe, and impress upon them the fact that it is to them, and to them alone, that we must look for acquiring the knowledge essential to the understanding of this subject, these teachers would do far more for the advancement of knowledge than they will ever do by their crude speculations and hasty deductions.

Up to the present blood-pressure observations have yielded but little information of real value.

It must be admitted that when we attempt to review the subject, very little of practical value has come out of the great amount of time and labour that have been spent on this subject. In place of the statement of the old physicians that the pulse was firm and hard we get now a definite and precise figure which most people imagine to be more scientific, as it is supposed to represent the systolic blood-pressure. In reality it does nothing of the sort, for the figure obtained by the use of the armlet differs from that obtained from the pressure taken directly from the artery. There is no doubt that it has some relation to the actual blood-pressure, and we can safely say that when we get a high figure or a low figure it represents a high or low blood-pressure.

One of the most curious features seen in the writings of those who use blood-pressure instruments is their trust in the figures they employ. They often take them as if they were a constant standard. Now the blood-

pressure may vary with exciting causes, just as the rate of the heart does, and too often temporary and incidental variations are not appreciated.

Perhaps the most striking instance of a lack of careful observation is seen in the reckoning of the blood-pressure in irregular action of the heart. I have never come across a writer who recognised that in the irregular heart of auricular fibrillation, for instance, it is, as a rule, utterly impossible to get a correct instrumental reading. Each beat varies in its strength, sometimes remarkably so, but writers will ignore this and give a precise figure, which cannot possibly bear any relation whatever to the actual condition. Even if they take the strongest or the weakest beat, or if they strike an average, the observation is absolutely untrustworthy.

Claims are now being made that the differences between the so-called systolic and so-called diastolic pressure afford important information. While no doubt there are considerable variations found, yet here again we are at a loss to account for them. To attribute this or that to the condition of the heart muscle, as is sometimes done, is to overlook the fact that the heart muscle only provides the force for maintaining the blood-pressure, the variations being dependent on the other factors of the circulation.

High blood-pressure is looked upon as in itself an object for treatment, and as the cause of high blood-pressure is unknown the treatment can only be haphazard.

It is as a guide to treatment that the blood-pressure is supposed to be of especial value. Happily, here we are prevented doing any harm, for we are practically powerless to affect it. Hot baths or some drugs, as the nitrites, may reduce the blood-pressure, but the effect lasts only a few seconds or as few minutes. By starvation and purging the blood-pressure may be temporarily reduced, but it soon returns to the

old height. It has been a matter of interest to me for many years to note the attitude of spa physicians towards blood-pressure. Each physician advocates the claims of his particular spa for the reduction of the blood-pressure. A few years ago the virtue was claimed for many spas that their waters produced a strong and firm pulse, and now, when a low blood-pressure is the fashion, these same waters have the contrary virtue and reduce a strong pulse to a weak one. It was a habit of mine for many years to take the blood-pressure of a patient who was about to proceed to a spa for treatment. When he returned he generally brought a letter from the spa physician stating that the patient had received great benefit, and that in particular the high blood-pressure had been greatly reduced. In every case where I took the blood-pressure on the patient's return it showed no difference. Some time ago I was consulted by a man on his way to a watering-place. His blood-pressure was 190 mm. Hg. He returned two months later, very cheerful because his spa physician had reduced his blood-pressure to 130 mm. Hg. I again took the blood-pressure and found it 195. I did not undeceive him, as the delusion made him very happy.

I have been consulted by a great many people because of their high blood-pressure. Most of them have been told of the height their blood-pressure has reached, and they have willingly submitted themselves to treatment, many of them going from one authority to another, and with each change undergoing a new treatment. It has been a matter of wonder to me to see the extraordinary resourcefulness of those who treat blood-pressure and the numerous theories that guide them. The drugs that have been employed are legion in number, diets of the most contrary descriptions—all-flesh diet, vegetarian diet, purin-free diet, and various other modifications; baths and waters and exercises of all kinds; high frequency current and other electrical devices. Notwithstanding this the blood-pressure has remained high.

Blood-pressure should never of itself form the basis of an opinion.

Up to the present no definite principle of the interpretation of blood-pressure records has been determined. Personally, when I find a systolic blood-pressure approaching 200 mm. Hg., I seek for evidence of damage in the blood-vessels and in the heart and in the kidneys, and it is upon the results found there that I base both my prognosis and treatment. In fact, in all circumstances, the blood-pressure, like murmurs, should not form the basis of an opinion, but should be one factor and that not the essential.

How blood-pressure research should be prosecuted.

In treating the subject in a somewhat disparaging manner, it is not my intention to discourage the use of blood-pressure observations. Far from it. My intention is to insist upon the importance of investigating the matter on the lines that will be productive of useful information, and I write this so that some of the fallacies and short cuts that at present are hindering the pursuit of a very important subject may be avoided.

I would suggest that general practitioners should take note of the blood-pressure in a number of their patients, particularly those they are able to keep under observation. These observations should be taken in the young as well as in the elderly. They need not at first be taken frequently, but as circumstances permit. From time to time certain cases will be found which need more careful observation, and these should be persistently followed. In every case attendant circumstances should be noted, particularly the state of the arteries, heart, and kidneys. It is only by approaching the problem in this way that the condition can be observed in its proper perspective, that the circumstances which favour its onset and modify its progress can be recognised, and the factors which influence its bearing on prognosis be accurately defined.

Blood-pressure instruments.

There are numerous instruments to be had. A very useful one is Martin's modification of the Riva Rocci. The observation should be made by the auscultation method. This is done by listening with a binaural stethoscope at the bend of the elbow over the artery. Air is pumped into the armlet, which surrounds the upper arm, till the pressure stops the flow of blood in the artery below. The air is then slowly allowed to escape and the eye watches the fall of the mercury in the manometer. As soon as a little blood escapes beyond the armlet a soft sound will be heard in the artery, and usually at the same time a faint oscillation with each beat of the heart will be seen in the mercurial column. The height of the mercurial column is called the systolic pressure. As the air escapes and the pressure falls the sound in the artery becomes clear and distinct. This clear sound is heard till the pressure gets very low, when it ceases. Before it ceases the sound becomes muffled, and it is the height of the mercurial column at this stage that MacWilliam suggests should be taken as representing the diastolic pressure.

CHAPTER XVIII

PROGNOSIS

THE principles of prognosis have been so fully dealt with already that here I need only summarise them, and indicate, by a few instances, how they may be applied.

The guiding principle on which a prognosis should be based is an appreciation of the functional efficiency of the heart muscle, and its ability to overcome any embarrassment to its work.

It is from this standpoint that every form of disease—valvular, myocardial, and arterial—has to be gauged. The data necessary for the forming of an opinion are present in each individual, though we may be unable to appreciate them. It is for this reason that I have insisted, all through the present work, on the imperative necessity of a due appreciation of the relation of every sign and symptom to the efficiency of the heart. Moreover, it is to be clearly recognised that each case requires special study. The fact that the physical signs and the patient's sensations in any particular instance might seem to indicate that the case belongs to a common type is no excuse for want of special study. The personal element and the patient's daily routine enter largely into the composition of the disease-picture. It is manifest, for example, that an impaired heart in a man who can lead a life of leisure has a significance different from that of an impaired heart in a man compelled to lead a strenuous and fatiguing life.

Not infrequently we may detect signs regarding the nature of which we are ignorant. Nevertheless we have

to estimate the bearing of these signs on the patient's future. We may find, on testing the functional efficiency of the heart, that the organ is impaired. The question we have to answer then presents itself to us in this way: Is the heart muscle capable of maintaining an efficient circulation, even though its field of response is limited? It is well known that many people with damaged hearts and a limited field of response reach an old age and show no signs of heart failure. This fact leads us to a consideration of the question whether the limitation is stationary or progressive. If it is stationary we may say that the outlook is favourable, so long as the individual is capable of living within his limitations. If, however, the field of response is gradually becoming more restricted, we must then have regard to the rate of progression of the limitation, to the possibility that rest may restore the heart weakness, and finally, to the ability of the individual to live a more restricted life. The results of our study of these factors must be our guides in giving a prognosis.

This applies to those cases in which the rest capacity of the heart is not impaired and in which the heart weakness is shown only by a limitation of the reserve energy. When there are objective signs of heart failure, such as dropsy, enlarged liver, and orthopnoea, and when these signs cannot be removed, the patient's future may be reckoned in months.

When symptoms of heart failure are present prognosis should be delayed until the effects of treatment are recognised.

In most cases exhaustion of the heart, with its varied symptoms of inefficiency, arises because the patient is unaware of the existence of impairment and so forces a damaged organ to do the work which is usually demanded of a healthy one. This is the reason why we see so often a gradual exhaustion of the reserve capacity, with the appearance of the characteristic symptoms which I have already set forth. The process of exhaustion may have

been going on for years before the case is met with, the early signs of inefficiency having been ignored until some sign has compelled attention. This is particularly the case with patients whose hearts have become impaired by the changes associated with advancing years—those patients, for instance, who ultimately exhibit signs of angina pectoris. As I have pointed out in the chapter on angina pectoris, it may not be possible in the first instance to say whether the exhaustion which produces the pain is due to a heart already slightly impaired and now exhausted by prolonged over-exertion, or to an advancing disease. A period of judicious treatment clears the matter up; for while, in the former case, a remarkable degree of recovery may follow rest, in the latter, in spite of all forms of treatment, recovery is slight. The same principles guide us in regard to other evidences of heart failure—*e.g.*, dropsy. It happens not infrequently that a slightly damaged heart in the young (*e.g.*, as a result of rheumatic fever) is not recognised until heart failure occurs. With rest and appropriate treatment recovery may, in this type of case, be so complete that the patient may be able afterwards to follow a strenuous occupation. It may be put down as an axiom that all hearts with a normal rhythm, in which there are present slowly advancing changes, recover from their first attack of failure if rested. The recovery may, however, be limited in degree where severe damage is present. The reason is easily grasped when the manner of heart failure is appreciated. Heart failure being due to a forcing of the organ to perform more work than it is fit for, reduction of effort and rest allow a measure of the lost strength to be regained.

The factors which have to be weighed, then, in forming a prognosis are the extent of recovery after treatment, the prospect of the patient being able to lead a useful life within the limits of his strength, and finally the progressive or stationary nature of the disease.

I have already dwelt on the importance of treatment as a factor in prognosis in auricular fibrillation.

No attempt should be made to base the prognosis on the presence of only one abnormal sign.

It may be thought that insistence on such a statement is not necessary, but experience has shown that in practice no obvious rule is so persistently neglected. It would not be too much to say that the vast majority of members of the profession have failed to grasp the true inwardness of what is conveyed in this statement. I have frequently seen individuals rejected for the Army and for life insurance and subjected to prolonged restraint and utterly unnecessary treatment because of the failure of their medical advisers to comprehend this principle. I have, in dealing with the more common physical signs, such as murmurs and irregularities, endeavoured to lay down the principles which should be a guide in estimating their value. These views I have repeatedly expressed; and over and over again I have been met with the assertion that they were perfectly understood. Nevertheless, among all classes of the profession, the old and experienced as well as the young fresh from schools, and even among teachers themselves, I find that, although the truth of the statement is admitted in theory, in practice it is ignored. I have already illustrated the foolishness of this procedure when speaking of the extra-systole, and the vague fears that are often aroused in the patient's mind when the doctor hints at an irregular action of the heart.

In like manner the presence of a murmur is frequently taken as a sign that persistent treatment is necessary. All these futile attempts are made because, in practice, the prognostic significance of simple phenomena has never been understood.

We see, therefore, that the formation of a prognosis is of great importance in the everyday life of the general practitioner as an adjunct to treatment. I insist *that no serious view of a case, far less a grave prognosis, should be based on the presence of one physical sign.* It follows, then, that in any given case which presents itself

for treatment, one must closely study the nature of the patient's sensations, and must be able to apprehend the significance of any abnormal sign which may be detected. I deal with this matter in speaking of treatment, but there are occasions other than mere therapeutic ones when it is necessary to have a clear idea regarding the future of the patient. The mode of life a patient should follow and the occupation he may engage in will often have to be determined. As it is impossible to lay down special rules for every case, a full comprehension of the manner in which heart failure is brought about becomes very essential, since from this comprehension only can a principle be obtained which will suffice as a guide. There are a few special applications of this principle which suggest themselves, which may be of value when in doubt, or in the difficult situations to which they refer.

Prognosis and pregnancy.

To every general practitioner at one time or another the question will be put whether a woman with a heart affection should be allowed to run the risk of child-bearing. He will have to decide the question. For many years I devoted much time to finding out the changes in the maternal circulation in the healthy and in those with diseased hearts. I found that many women, who attended to their household duties and came through their confinements unscathed, became exceedingly short of breath and had dropsy during the later months of pregnancy. Many even with organic heart troubles were little the worse for their pregnancy. Those in whom the pregnancy caused serious heart failure were chiefly cases with mitral stenosis. As this condition is frequent in women it requires special consideration in relation to pregnancy. I have had a few cases of women with mitral stenosis who suffered little or no discomfort during child-bearing. These were old-standing cases, however, and the disease was either stationary or was advancing so slowly that for years I

could detect no change in the character of the murmurs. There was present, moreover, only a presystolic murmur; the heart was not enlarged, or was but very slightly enlarged, and the response to effort was good. In those patients who suffered from heart failure during the pregnancy the disease was either of more recent origin—*i.e.*, a shorter period had elapsed since the causative attack of rheumatic fever—or it had progressed till a diastolic murmur was audible as well as a presystolic, and the heart response to effort had become distinctly impaired. The heart failure in these cases became extreme before full time, or an extreme condition of heart failure occurred within ten days of the confinement. Some of the women died; others recovered, but showed permanent impairment of the heart. In some of these women, who came through their confinements fairly well, the heart regained but a limited amount of strength. In cases of auricular fibrillation the same experience was met with. Even when the pregnancy and confinement were well borne the patient's strength was permanently weakened; in other instances the result was actually disastrous. There is no difficulty in understanding this state of matters when we consider that in most, if not in all, of these hearts mitral stenosis was present and was adding to the embarrassment of the organ.

I have only met with two cases of pregnancy in which there was aortic regurgitation. Both women became very weak and ill towards the end of the pregnancy; both bore their confinements well; yet both gradually got weaker and died some months after.

My experience would therefore suggest that pregnancy should be forbidden in young subjects with progressive mitral stenosis (see Chapter X), with auricular fibrillation or with aortic regurgitation, unless the leakage is slight and the left ventricle not enlarged.

Prognosis and anæsthesia.

Another question that will frequently arise is whether a patient with some form of heart trouble will be able to stand an anæsthetic. My experience is limited to the use of chloroform, which I used freely in practice for over a quarter of a century. In the administration of the anæsthetic I was guided by the physiological effect on the consciousness of the patient. Thanks to careful watching I had few misadventures, and I am disposed to think that the changes that have been made in the methods of inducing anæsthesia are not all for the better. The fact that chloroform is falling into disuse confirms me in this view. It is a curious thing that, while in some hospitals there are no deaths in 20,000 cases of chloroform anæsthesia, in others there is a death in every two or three thousand. This would seem to suggest some fault in the administration rather than in the drug administered; an example of the same kind of fault is furnished by the frequent misuse of digitalis.

The data which guide me in replying to the question whether or not a patient should have an anæsthetic are obtained exclusively by consideration of the manner in which the heart maintains the circulation. If there is a good or fair response to effort I unhesitatingly give consent to the use of the anæsthetic, no matter what the organic lesion may be. In cases of extreme exhaustion of the heart through long-standing disease, as, for example, when angina pectoris is easily provoked; when there is marked limitation of the field of response—*e.g.*, with auricular fibrillation; when the patients are fat, wheezy people with liability to bronchitis, the anæsthetic should be given only if absolutely necessary, and then with the greatest care.

I have successfully given chloroform in cases with very marked heart-disease (complete heart-block, auricular fibrillation, and all forms of cardiac disease), and I am disposed to believe that the guide should always

be the state of the heart muscle and its efficiency as found out by its response to effort.

The cause of sudden death was stated by MacWilliam long ago to be, in all probability, fibrillation of the ventricles. MacWilliam and Levy have since then, as the result of experiment, found that fibrillation of the ventricles is also the cause of sudden death under chloroform anæsthesia, and the latter observer has stated that sudden death is more likely to occur with insufficient anæsthesia than with full anæsthesia—a conclusion certainly in agreement with the teaching Lister gave us.

Prognosis and congenital heart disease.

I have rarely been able to satisfy myself as to the real defect in congenital affections of the heart. And though I have seen a good many cases and watched individuals for years I have never been able to correlate the symptoms present during life with the post-mortem findings. As, however, my opinion is often asked, I give a prognosis upon the principles already laid down—*i.e.*, I consider the response to effort and the size of the heart. Many of the cases have lesions, such as a patent ductus arteriosus, or a loud, rough, systolic murmur, with no increase in the size of the heart. In those the outlook is good. But when the heart is much enlarged and its efficiency markedly impaired, when cyanosis is readily provoked by crying or by effort, the outlook is not good. A crippled existence for a few years, ended by some intercurrent disease, is all that can be anticipated.

In some cases murmurs heard at birth may disappear entirely as the child grows; in these cases the heart appears to be normal in all respects.

In other cases, which have no marked changes in the heart, the body development is poor. It is probable that the congenital condition here is the narrowing of an orifice; instead of this orifice becoming increased in size as the body develops, it remains unwidened, and so

limits the distribution of the blood to the body. The body is thereby weakened and so becomes liable to the invasion of disease, especially of tubercle.

Nevertheless, there may occur a wonderful modification of other structures to make good the defect. This is seen in that remarkable condition in which the aorta becomes obliterated (coarctation of the aorta). I have only seen one case—a middle-aged woman; in this case the peripheral arteries had all become greatly enlarged, so that the nourishment of the trunk and limb was quite normal.

The personal element in prognosis.

There is one factor which must be taken into account in estimating the value of a prognosis—the personality of the observer.

I have two friends—both very distinguished physicians—who illustrate in their own persons, in their temperaments, and in their attitudes towards their patients two great extremes. The one is buoyant and hopeful, and he confided to me that when in doubt he always gives weight to the most cheerful outlook. Even when there is a strong suspicion of malignant disease the patient leaves him buoyed up with hope. The other takes a gloomy view of everything that he does not understand. A sign of obscure origin is to him filled with the most appalling possibilities, and though he may say nothing to the patient, yet his doleful countenance and the solemn shakes of his head convey far more despair than would the utterance of the most hopeless prognosis. Modifications of these two types will be met with everywhere, and the prognosis is invariably biassed by their temperaments.

Mystery and prognosis.

If, then, we would guard against errors in our pursuit of a sound prognosis, we must realise and appreciate certain human characteristics which modify the interpretation of observations. We must remember also

that in drawing conclusions with a view to what, after all, is a form of prophecy, the prophet includes a good deal of his own personality. Again, there is one factor which influences all humanity, and that is mystery. Mystery not only influences the mind, but it influences it in a definite manner, by engendering fear. That which is mysterious is, unconsciously, assumed to be evil; the gloom which so often surrounds the prognosis in heart affections may be traced to this human peculiarity. If a metaphysician took the trouble to study the psychology of the educated and scientific man he would find a subject at once curious and interesting, and this more particularly if the scientific man happened to be exercised about his own inside. We are so accustomed to associate superstition with ignorance that it startles us to find in the educated evidence of the subjection of reason to faith. A highly-trained chemist will accept no statement about chemicals unless and until he has put that statement to the test. Let him, however, put some chemicals into his own stomach, by way of producing an effect upon disease, and at once he seems to become capable of believing that they are endowed with the most miraculous properties.

The heart is an organ so essential to life, and the mystery of death is so often associated with some of its disease manifestations, that any abnormal phenomenon is apt to cause apprehension in the minds both of the doctor and the patient, that this may be evidence of the thing that slays. I have frequently been called to see learned colleagues who have betaken themselves to bed because they have perceived an abnormal action of the heart—the same having no prognostic significance.

A prognosis based upon an intelligent perception of the meaning of signs or symptoms is the only means at our disposal for dispelling the depression produced by mystery. Some years ago a distinguished author consulted me; he was oppressed by the fear that he had got angina pectoris. As in the case of many intelligent

people, the name *angina pectoris* was surrounded in his mind by grave associations. His description of his sufferings, too, embellished by a very wide and artistic vocabulary, gave a tragic appearance to his complaint. A little time before he consulted me I had read one of his short tales in which supernatural elements were described so graphically as to produce the most terror-inspiring effect. After examining him I referred to this tale of his and said that he had not given his readers any clue to the nature of the supernatural events. He then discoursed to me on the art of mystery, and, among other things, said that so long as the real cause of fear was unknown, the human mind unconsciously assumed that what it could not understand must be fearsome. To draw aside the curtain was to banish the sense of terror. After he had finished I told him that in his own case this fear of the unknown was the source of his trouble. It was the mystery associated with the idea of *angina pectoris* that was making him ill. I then explained to him in simple language the nature of his trouble and sent him away much comforted and much happier; and, as years passed, he found that my prognosis was justified.

The guarded prognosis.

There are many conditions regarding which our knowledge is so limited that we are at a loss what to say and do in connection with them. There is, fortunately for us, a wonderful bridge by which we can escape from this difficulty, and that bridge is the guarded prognosis. When a patient presents an abnormal sign the significance of which the doctor has no idea, the latter says, as a rule, that the condition is an obscure one; that the patient must be careful that no injury results from an aggravation of the condition; as to the future, he must "play for safety." In the meantime he may employ some treatment in the hope that it may prevent something worse from happening.

By this means the physician frequently acquires the reputation of being a very safe man; but we may be forgiven for asking: What of the patient? The abnormal sign may be, often is, of no significance, yet the patient goes away carrying in his mind an ill-defined fear that something within him is amiss and that dreadful things may happen. Not infrequently his mode of life has to be altered, so that his business affairs are interfered with, and even his future career modified.

We must realise that a guarded prognosis is really a confession of ignorance, and that the real state of affairs is, that there is present a sign of which the physician is ignorant. As he will not admit his ignorance his patient is made to suffer.

I admit that our ignorance of so many complaints is so profound that there is nothing left for us but to give a guarded prognosis. But, in doing so, we should realise that it is our own knowledge that is at fault, and make it our daily endeavour to find out the real significance of that of which we are ignorant. If we come across some phenomenon of disease of which we have no experience we can, of course, do no better than give our guarded prognosis; but when a physician is constantly coming across the same phenomena for twenty or thirty years, and at the end of that time is still giving a guarded prognosis, it is evident that the real conception of what clinical medicine is has not entered into his mind.

Statistical methods in prognosis.

Attempts are made at times to estimate the probabilities of life by finding out at what age a great number of people die who, during life, have shown some sign, such as a murmur or an irregular action of the heart. It is curious that the fallacy of this view is not recognised. If I were to note the age of death in a great number of people with soft corns and were then to lay it down that the average period of life in these persons

represented the probable duration of life in all people with soft corns, the absurdity of this method of reasoning would be perceived at once. Why is it absurd? Because a soft corn is not a fatal disease. Now the vast majority of signs on which the insurance examiner bases his statistics are as free from risk to life as are soft corns, so that when the examiner takes a murmur as a criterion he is taking a sign the cause of which may have no bearing whatever on the individual's health. Those who have appreciated what I have said about heart failure will appreciate the truth of this criticism.

CHAPTER XIX

PRINCIPLES OF TREATMENT

In the past, lines of treatment have too often been adopted before guiding principles were understood

THERE is a widely-established and very ancient belief that for every form of complaint an appropriate remedy exists. In accordance with this belief remedies have been suggested during untold ages, so that we find records of energetic and resourceful treatment from the most ancient times to the present day, and among the most backward races as well as among the most advanced in civilisation. As treatment was employed in primitive times, when neither the nature of disease nor the principles on which it should be treated were understood, much of the therapy was based on empiric methods. The faith in certain of these old remedies has been handed down from generation to generation, and though no doubt faith has been modified from time to time and new beliefs added by each new generation, yet the empiric methods have not been displaced. If one looks back on the literature of the last hundred years one finds this process of addition in operation. Some drugs and methods have been employed for varying periods, and some have fallen into disuse, while others have been adopted in their place. Throughout all these changes no fundamental principles guided physicians in the adoption or rejection of a drug. The circumstance which has determined the adoption of a new method has been the apparent improvement of the individual in response to its application, backed by the authority of the physician who applied it. With the fading of the physician's reputation the method has,

as a rule, fallen into disuse, unless there has arisen in the meanwhile a financial interest in its continuation, in which case brazen advertisement may have kept it going for a time.

There is a great need for the intelligent study of methods of treatment.

This advocacy of remedial measures based on no definite principle is as marked at the present day as at any, even the most benighted, period. I have been struck by the criticisms passed on my suggestions with regard to treatment in diseases of the heart. The burden of these criticisms is that I recommend so few remedies. A great many drugs, baths, exercises, and other methods, the vast majority of which are entirely without effect, are recommended by writers. It is easy for a writer to mention this or that treatment; but critically to test a line of treatment requires such careful observation that few writers, even those who advocate their own particular methods, ever attempt it. It has therefore always been my aim in writing only to give the results of my own experiences. In employing methods of treatment I naturally have followed, in the first place, the injunctions of authorities who have prescribed a certain line. But I always followed my treatment critically and endeavoured to find out whether the line recommended had the result which had been described. This work has entailed an enormous number of observations and long periods of investigation. The result of it has been a growing conviction that the great bulk of the remedies and methods tested are without any real benefit. I have acquired a certain amount of knowledge which enables me to perceive clearly the object to be aimed at in any given line of treatment. If the advocates of any new method cannot show some reason why their method will attain this object, but instead base their recommendation on a fanciful concept, or on imperfect observation, I know,

beforehand, it will be a waste of time to investigate the matter further. Unfortunately many recommendations are made by people who have not acquired the knowledge of what the object of treatment really is, and who do not know the significance of many manifestations which the heart can exhibit.

The essential object in treatment is to anticipate and prevent heart failure, or to restore the heart if it has failed.

Before dealing with the matters essential to the treatment of heart affections it is necessary that we should hold clearly before us what it is that we attempt in our treatment. In order to emphasise this necessity I would refer again to the essential question which the doctor must put to himself when he detects something amiss with his patient's heart. The question he must ask himself is : " Is this sign an evidence of heart failure, or does its presence foreshadow the occurrence of that state ? " It is always heart failure that is to be feared, that if present has to be cured, and that if foreshadowed must be anticipated and prevented. If the doctor can satisfy himself that the sign present indicates neither the one state nor the other, then the fear of heart failure may be put on one side so far as treatment is concerned.

Another matter we must endeavour to recognise, namely, our own limitations. If we detect a lesion which is stationary, but which hampers the heart in its work, we know that heart failure may supervene, and our treatment will follow the lines I will shortly indicate. But having recognised the lesion we must also recognise how helpless we are in removing it. If, for instance, there is evidence of a valve defect, mitral or aortic, we must endeavour to find out whether or not the lesion is a progressive one. We know that a mitral defect may exist for long years and never embarrass the heart muscle nor show any sign of becoming worse. Damage to an aortic valve, again, may become stationary; the regurgitation embarrasses the left ventricle, but the ventricle may be

spared exhaustion if the patient lives at a lower level in regard to exertion. This, however, is not cure, and we know only too well that attempts to cure the valve lesion would be vain. In like manner, when we are face to face with a progressive lesion, our power of checking its progress is extremely limited. Thus, mitral stenosis is seldom a stationary disease; the stenosis slowly but steadily increases. The rate of increase varies and may be measured in a rough manner in the way already described. What we have to recognise is that we cannot stay the progress of the disease. In myocardial disease too often the changes are progressive and we are equally helpless. We observe how in some elderly people arterial and cardiac degeneration proceeds rapidly, while in others it is very slight and proceeds slowly; yet we are powerless to stay its progress. I know that this view is strongly resented by some, and that it is maintained that by dietetic, medicinal, and other means these changes may be stayed and even removed. As, however, these changes are fundamentally the same as those which produce the grey hairs and the attenuated skin of the elderly, we might as well—while using the various nostrums and treatments—expect signs of the staying of those senile changes as signs of the staying of the heart condition.

I mention this, not to discourage the doctor in his work, but in order that he may clearly distinguish between what is remediable and what is irremediable; when he is face to face with the irremediable he should recognise the fact, and thus make it possible to give such advice as will lead the patient to make the best of his condition and to avoid the impending danger of premature heart failure.

Before attempting treatment be assured that the symptoms are produced by an impaired heart.

I repeat that it must be clearly appreciated that the danger in all cases of heart affection is the occurrence

of heart failure, and that treatment must be directed to preventing the occurrence of that condition, or to the restoration of the heart if it has already begun to fail.

Before attempting treatment one must be certain that the symptoms of which the patient complains—for example, weakness, breathlessness, and so forth—are primarily cardiac in origin, and not due to some general cause, like infections, anæmia, or diseases of other organs. The distinction between such symptoms as exhaustion and syncope and those which are indicative of cardiac failure must be clearly comprehended.

The essential principle in treatment is the care of the heart muscle.

When the evidence is conclusive that the symptoms are due to cardiac failure it may be taken for granted that the failure has been brought about by the heart being forced to do more than it was fit for. The first indication, therefore, is to remove all conditions that prevent the heart from getting sufficient rest; among these conditions are over-work, sleeplessness, worry, and digestive troubles. Where it is necessary for the patient to pursue his occupation, his daily routine must be fully considered and all forms of effort eliminated except those which are necessary for his occupation. There are innumerable little ways in which this lightening of effort can be carried out, and the careful discussion of these, when the patient understands the cause of his weakness, will often be of the greatest value. This is a point on which I lay the greatest stress, for frequently I see business and also working men rendered more ill and made miserable by enforced rest, when a mere easing of the daily work would have proved—and in the end did prove—amply sufficient.

When the necessary daily work is so strenuous that the relief obtained by restriction of effort is but temporary, it is better to advise the patient to change his

occupation. This applies especially in the case of the young. Indeed, in the case of young people with damaged hearts the selection of an occupation which does not entail severe bodily labour may be the means of prolonging life and enabling the cripple to pursue a useful existence. One must, of course, be certain that the damage is of the kind which really demands such restrictions, for very often I find the young restricted in their activities on account of signs and phenomena which indicate no impairment of the heart.

CHAPTER XX

TREATMENT (*continued*)

REST, DIET, EXERCISE, BATHS

Rest.

WHEN cardiac exhaustion has gone so far that breathlessness or pain is easily induced, and objective signs, such as dropsy, are present, rest is imperative. While other remedies may be necessary, rest is the most potent remedy of all.

In these extreme cases it is not always easy to decide what position the body should occupy so that the greatest benefit may be secured. As a rule, the patient's sensations are a very good guide. It may be that lying in bed causes such distress that the patient is not at ease. In these cases he assumes some position which he finds removes the discomfort, such as sitting in a chair or leaning forward on a support. These individuals should be permitted to assume the position in which they find the most comfort, or, rather, the least discomfort, as that position favours the circulation in regions which induce the distress—*i.e.*, in the lungs and brain—though it may militate against the circulation in other regions—*e.g.*, the legs. When dropsy tends to increase in these cases, careful change of position, raising the legs as high as possible, or deft bandaging and massage, may do much to diminish the swelling, other treatment being, at the same time, directed to the dropsy and heart condition. When the failure is not so extreme, and particularly if there is dropsy, complete rest in bed is of the greatest service, and many cases recover without other treatment. If necessary,

the shoulders may be raised, by means of pillows or a bed-rest, to such a height as may prevent attacks of dyspnœa from coming on during sleep. In all cases, mild or severe, every source of discomfort arising in other parts of the body should be attended to—*e.g.*, an irritating skin affection, piles, and frequent micturition.

Diet.

Digestive derangements often disturb a damaged heart and provoke abnormal action in fairly sound hearts. This disturbance is often due to flatulence or fermentation in the stomach and bowels. The best treatment of the condition in my experience is the giving of the patient's food in a very dry state. Butter on bread or toast should be forbidden, and the mouthfuls of dry bread should be small and should be so thoroughly masticated that the food is moistened to the consistency of cream by the saliva alone. The meals in all cases should be small, but should be taken at frequent intervals; only small quantities of fluid (tea or coffee) should be permitted after the meal, though between meals more fluid can be permitted if needed. I do not restrict my patients to any one kind of food, but prefer to leave the selection to the patient's intelligence, advising him merely to avoid what does not agree with him. I find that there are few articles which do disagree with these people if this plan of thorough mastication is followed, and all moist and sloppy foods are avoided (thick soups, milk puddings, porridge and milk, Benger's food, etc.). Even in extreme cases this method of dieting is of the greatest value, as in those confined to bed abdominal distension from flatulence, aggravated by the ingestion of fluids, is often a distressing complication.

Exercise.

When the patient is fit to go about he should be encouraged to take exercise in the fresh air. The

amount of exercise he may take will depend upon his strength, and this strength can be gauged by his sensations. So long as a man who is taking exercise remains free from distress or discomfort, the exercise is doing him good. The principles underlying this statement will be realised if the nature and mechanism of heart failure has been clearly comprehended. We get, indeed, in this statement a guide which will serve us in all cases, and we may lay it down that the patient should diminish or cease his efforts on the first appearance of a distressful sensation.

It is far better that the patient should take his exercise in the fresh air, in some form congenial to his temperament and suitable to his strength, than that he should take it by making systematised movements, or undergoing what is called graduated exercise. To every case of heart affection there is a mental side; mechanical exercises tend to remind the patient that he is an invalid, whereas a game, or an exercise in the fresh air with an object beyond the consideration of health, has a beneficial mental effect. The practice of allowing a patient to take that amount of exercise which gives pleasure and does not give rise to sensations of distress or discomfort, is based upon principles of the highest importance. If the mode of onset of heart failure and the symptoms by which it is revealed are borne in mind, the significance of this principle will be at once apparent; for I pointed out that exhaustion of the heart muscle showed itself, in the first instance, by producing sensations of distress.

Moreover, as all organs benefit by the judicious exercise of their functions, so likewise the heart is kept in good condition by the exercise of its functions, so long as the exercise does not produce exhaustion. This truth has been clearly perceived, and a great many forms of exercise have been evolved. On behalf of some of these—the resistance exercises of Schott, for example—fantastic claims have been made. Other methods, such

as the graduated exercises of Oertel, have been found useful; but these exercises are not based on a proper recognition of the object to be aimed at. The object to be aimed at is the strengthening of the heart muscle. Now, by summarily laying down that so many yards should be walked one day and so many another day, the physician shows that he has failed to take into account the peculiar nature of the heart functions. Thus the power of response of the heart to effort varies greatly in the same individual from time to time—one day a patient with an impaired heart can undertake a good deal of effort with comparative comfort, whereas on other days the same amount of effort causes him distress. This is due to the fact that the heart may be embarrassed by a variety of conditions, such as gastric and intestinal disturbances, want of sleep, and the state of the weather. The patient's sensations are therefore a valuable guide and indicate the amount of effort which can be undertaken by him with safety in all circumstances.

Baths.

The immersion of the body in water at different temperatures has a great effect upon the peripheral circulation and also modifies the rate of the heart. Where the temperature of the bath is above that of the body, the peripheral vessels dilate and the heart's rate increases; when it is beneath that of the body the peripheral vessels contract and the heart's rate diminishes. It is doubtful whether these varied reactions have any permanent effect on the heart. That a warm bath may afford temporary relief in special circumstances may, however, be quite true,

Great claims have been advanced on behalf of the "magical effect" of the carbonic acid baths of Nauheim. Anyone with an elementary knowledge of physiology must, one would suppose, recognise that the immersing of the body in water containing carbonic acid is not likely to produce an effect on the heart. The carbonic

acid simply cannot reach the heart. A body is immersed in the waters of Nauheim when these waters are at a lower temperature than the body. The heart's rate naturally becomes slower; yet the slowing is described as a remarkable effect of the Nauheim waters. If anyone will test the effect of a bath of ordinary tap water, taken at a lower temperature than the body temperature, he will discover that these effects are identical with the effects produced by Nauheim baths. When the warm body lies in cooler water saturated with carbonic acid small globules of the gas are liberated which adhere to the skin and the skin becomes red. This simple phenomenon is taken as representing a remarkable effect on the peripheral vessels, and is supposed to affect the heart in some mysterious way. Contact with many patients who visit Nauheim soon reveals the truth that what benefits do arise from this bath treatment come, not from a specific virtue residing in the waters, but from the restful, peaceful atmosphere of the place and from the stimulation of great faith in the efficacy of the cure. The absurdity of the claims advanced on behalf of Nauheim is at once apparent when it is realised that all varieties of cardiac symptoms are treated there indiscriminately and upon the same lines. The reputation of Nauheim baths and of the artificial imitations of them depends more on brazen advertisements, financial interests, and the slavish tributes of other spas than on an intelligent appreciation and application of the principles of treatment,

CHAPTER XXI

TREATMENT (*continued*)

DIGITALIS, STROPHANTHUS, THE NITRITES, SEDATIVES,
OXYGEN, ATROPINE, OTHER DRUGS

Digitalis.

SINCE Withering published his book on Digitalis in 1745, this drug has been recognised as a valuable remedy. Originally it was prized as a diuretic, afterwards it became better known still on account of its effect upon the heart. Yet, enormous as has been the amount of research in laboratory and hospital ward directed to elucidating the properties of the drug, we are still, at this day, far from appreciating its effects and in recognising its proper sphere in treatment. In the following remarks on the subject I give the results of my own experiences, and I acknowledge that these are, of necessity, limited; for this is a subject which no one individual can hope effectually to elucidate. My reasons for limiting myself mainly to my own experiences are that the subject has hitherto been so confused that no clear principle has ever been laid down to guide the practitioner in the employment of this valuable remedy. This has been due, in the past, to the fact that the mechanism of the peculiar rhythms of the heart and the signs of disease of the organ have not been clearly recognised. At the present day we are in a better position, though there is still much to be learnt. Moreover, the experimental pharmacologist, to whom we have looked for guidance, has been at a disadvantage which he has never appreciated, in that his experiments have been made with large doses in order

to get rapid results—doses that are quite different from the medicinal doses we give to our patients. Further, the animals experimented on by the pharmacologist have healthy hearts and blood vessels, whereas in our patients these organs are diseased; one of the most striking results that have come out of my investigations is that the action of digitalis on the human heart depends in a great measure on the nature of the disease present in the heart, and this is true especially of the cases in which digitalis is most beneficial. It follows, therefore, that the laboratory worker is shut off from the study of the drug in these very fields of therapy where it is most needed.

When I was engaged in general practice I kept fairly careful records of the cases to whom I gave digitalis. After ten years I gathered all my notes together and analysed them, and there came to light a remarkable variety of effects. In a large number of cases no effect on the heart could be detected, even when the drug had been persevered with so far as to produce sickness and vomiting. In other cases remarkable effects were shown on the rate and rhythm of the heart, and in some there were the most beneficial effects on the patient's condition. At that time I was engaged in the study of abnormal rhythms of the heart, and I found that digitalis in some cases produced extrasystoles, heart-block, the pulsus alternans, and even auricular fibrillation, besides other peculiar effects which even to-day I do not fully understand. The most striking effects occurred in hearts which were manifestly much damaged by disease.

Digitalis in auricular fibrillation.

When I came to classify the cases which had benefited greatly I found that the vast majority of them fell into one group. This group consisted for the most part of persons with advanced heart failure and an increased rate of contraction, sometimes as rapid as 140 or 150

beats per minute. In addition to the manifest relief of the patient obtained by the use of the drug there was a remarkable slowing of the pulse. With the fall of the pulse-rate to 70 or under the objective signs of heart failure, enlargement of the liver, dropsy, and orthopnoea usually disappeared. In every one of these cases the heart, before treatment, was irregular in its action, and showed that peculiar form of irregularity which I had first associated with paralysis of the auricle, and then, later, called the "nodal rhythm," but which we now recognise as "auricular fibrillation." I made a patient search through the literature to determine the kind of cases which had been found by other observers to benefit from digitalis. In most instances no notes of individual cases were given by writers; the vast majority of writers, indeed, stated their opinions so vaguely that no clear conceptions could be obtained. In several cases, however, references to the heart's rate and rhythm made it clear that the patients who obtained marked benefit were the subjects of auricular fibrillation.

During the time I had charge of the cardiac wards in the Mount Vernon and London Hospitals the effect of digitalis on the human heart was one of the subjects of research. My fellow-workers in these hospitals and I were able to add a good deal of more precise knowledge, yet the results we have obtained have been, on the whole, but confirmatory of those results which I myself obtained in general practice. I mention this to impress upon you how great a power of adding to our knowledge, even in therapeutics, the general practitioner possesses. I may go farther and say that in this field of therapeutics his services are absolutely essential, chiefly for the reason that when treatment has to be continued uninterruptedly for years, he is the only person who can follow treatment intelligently.

One of the results of my observations was the ability, not only to restore hearts when the failure was extreme, but to prevent the onset of heart failure. I was thus

able, by the intelligent use of digitalis, to ensure for many of my cardiac cases the comfortable and secure pursuit of their work.

Extreme heart failure is so frequently associated with, and induced by, auricular fibrillation, that the efficacy of digitalis is best shown in this condition. When it is understood that auricular fibrillation is present in about 60 to 70 per cent. of cases which suffer from heart failure with dropsy, and that the heart failure is induced or aggravated by its presence, the importance of appropriate treatment becomes at once evident. Every practitioner should, therefore, be familiar with the method and principles of the treatment



FIG. 20.

Rapid, irregular pulse, 140–150 per minute, due to auricular fibrillation with extreme heart failure.

of auricular fibrillation. When a great number of cases of auricular fibrillation are studied it will be found that, in the earlier days of the presence of this condition, the heart's efficiency varies considerably. In some cases there is but the slightest impairment with no objective sign, in others the limitation is considerable, while in others the failure may be extreme, with dropsy, enlarged liver, and orthopnoea. In nearly all the cases with moderate or extreme failure the heart's rate will be found to be greatly increased, even up to 140 or 150 beats per minute. Frequently the beats are so weak that many of them are not perceptible at the wrist (Fig. 20).

I am disposed to think that the heart failure is due to exhaustion of the heart muscle induced by the rapid rate at which beat follows beat. There is not, in fact, sufficient time for recovery between the beats. The out-

put is diminished and the surplus blood increases the embarrassment of the ventricle.

There may be other factors at work in producing the heart failure, but it remains true that any method which diminishes the rate at once improves the condition and enables the heart to recover. Digitalis effects this purpose, and we need but note how the fall in rate is at once accompanied by a surprising recovery, to perceive the relationship existing between these two circumstances. This, of course, applies to the earlier stage of the trouble. It is manifest that in time the best of hearts gives way and that, in the end, all remedies necessarily fail. In the vast majority of cases many



FIG. 21.

Slow, irregular pulse, 55-60 per minute. From the same patient as gave Fig. 20, 10 days later, after treatment by digitalis. In addition to the slowing of the pulse there was marked improvement in the patient's condition, though the irregularity shows that the auricular fibrillation persisted.

years of careful life free from heart failure can, however, be led when digitalis is properly administered.

When the rate is moderate—80 to 100 beats per minute, the effect of exercise should always be noted. In most instances, although the rate at rest is moderate, a great increase in rate, up to 130 beats per minute or over, takes place when exercise is undertaken.

In these cases digitalis is of service in controlling the rate during exercise, and should be administered according to the method described.

When the rate is moderate—70 to 90 beats per minute—with no disproportionate increase in rate in exercise, and the heart's efficiency is good, no treatment is necessary, and the patient should merely be cautioned against prolonged physical effort.

When there is marked breathlessness on exertion, or dropsy with an increased rate, the digitalis should be persevered with till distinct evidence of its effect is produced. This evidence may be shown first by a marked decrease in the rate (Fig. 21). In some cases, at the same time, nausea or vomiting occurs, while others suffer from a disagreeable oppression across the chest. Diarrhœa and headache occur in a few cases.

As soon as any of these signs appear the drug should be stopped, and should not be resumed until they have disappeared. This disappearance generally takes place in a couple of days after the first sign of a sufficiency, and then the patient usually feels extremely well. If the pulse-rate begins to increase again the drug should be given in half doses, and increased or diminished according to the reaction. In the majority of cases the smaller dose does not induce the disagreeable effect, but keeps the rate of the heart in check, so that often we can increase or diminish the rate by withholding the drug or by increasing the dose. In some cases five minims of the tincture, once a day, will keep up this control, in others it may be necessary to use double or treble this amount. In all cases it is incumbent on the doctor, in collaboration with the patient, to find the exact quantity of the drug that will keep the heart's rate in the most favourable condition for exertion, and this quantity continued while the patient lives.

Any preparation of the drug can be used, for no advantage appears to attend the use of special forms. I have never found the B.P. tincture fail me, and it is this form which I have used most frequently, because it is the cheapest, and therefore is within reach of the poorest. I begin usually by giving the patient one drachm per day—twenty minims taken three times a day—and I commonly get the result on the fourth to the seventh day. Sometimes the result is delayed a few days longer. An elegant and effective preparation is Nativelle's granules of digitalin—the white kind con-

taining $\frac{1}{240}$ of a grain. I have repeatedly compared the effect of these granules with that of the tincture, and I find that one of the granules has the same effect as fifteen minims of the tincture; therefore, when using the granules, four of them should be taken per day at the beginning of the treatment. If any of the other numerous preparations is used, the drug should be given in sufficient doses to produce an effect, the dosage being regulated by the principles I have enunciated.

Digitalis in auricular flutter.

In the condition allied to auricular fibrillation—auricular flutter—the action of digitalis is sometimes wonderfully beneficial. As I have already pointed out, the manifestations of this rhythm of the heart are varied and confusing; the majority of the cases, however, have a regular pulse-rate of from 120 to 160 beats per minute, while the auricles beat at double this rate. This rapid rate affects different hearts differently. In some it speedily exhausts the ventricles, and dilatation and extreme heart failure appear; in others, no sign of failure, except a slight limitation of the heart's response to effort is noticed.

The treatment of auricular flutter consists of rest and the administration of digitalis in the manner described as being necessary in auricular fibrillation. The drug must be given in full doses at first, and then, with the decrease in the pulse-rate, or on the appearance of nausea, it must be stopped. In some cases the digitalis causes the flutter to pass into fibrillation with a coincident great slowing of the pulse; when the drug is stopped the heart may resume its normal rhythm (see Figs. 22, 23, 24). In many cases, however, all that can be done is to increase the block between the auricles and ventricles, so that the ventricular rate decreases. By watching the effect of the drug closely we may do a good deal to help the patient, even when the drug has only this temporary

effect. I must confess, however, that the treatment of many of these cases has proved very unsatisfactory.

Other conditions in which digitalis is beneficial.

While it is in auricular flutter and fibrillation that the most dramatic effects of digitalis are perceived, there is a widespread belief that the drug is of use in every

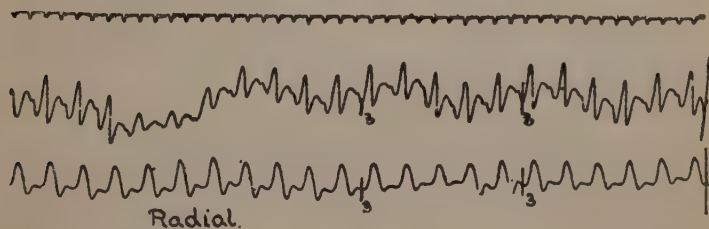


FIG. 22.

Tracing of radial and jugular pulses during a period of continuous tachycardia due to auricular flutter. An electro-cardiogram showed that the auricle was beating at the rate of 280 beats per minute, while the ventricular rate was 140.

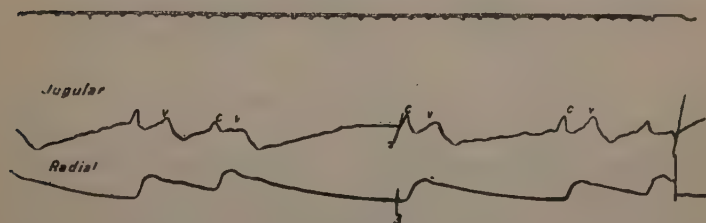


FIG. 23.

From the same patient as Fig. 22, after digitalis, which had changed the auricular flutter into auricular fibrillation. The slow, irregular pulse and the characteristic jugular pulse indicate the presence of auricular fibrillation.

form of heart trouble, or in any form of illness in which the heart is supposed to be weak. A careful inquiry reveals the fact that there are but few other conditions which are improved by digitalis. In all cases of heart failure with dropsy digitalis should be tried. It is probable that the beneficial effect produced by the drug is due to its diuretic effect rather than to its action on

the heart. In many cases with a normal rhythm the improvement in the patient's condition with the disappearance of the dropsy is accompanied by no appreciable change in the heart. In working out the relation of the action of digitalis to the heart and kidney, we have not reached a satisfactory conclusion. In some of our cases of auricular fibrillation the diuresis occurred within forty-eight hours of the administration of the drug, whereas no effect on the heart was perceptible until several days later. Moreover, the kidney functions,

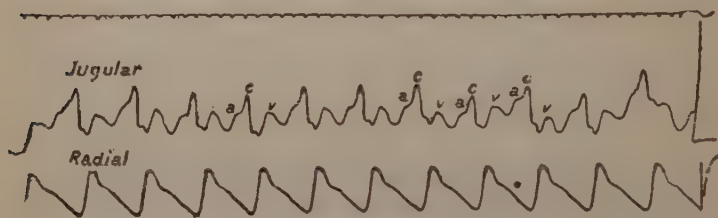


FIG. 24.

From the same patient as Figs. 22 and 23. After the digitalis was stopped the heart-beat resumed the normal rhythm as shown in this tracing.

when there is dropsy, are peculiarly unstable, and at times some very slight cause may produce abundant diuresis. Thus many doctors will dilate upon the marvellous character of some "gunshot prescription" (or purveyors of drugs will tell of the wonderful diuretic effects of some special preparation), because in one or two instances abundant diuresis has followed the administration of the nostrum. I remember on one occasion prescribing the well-known pill of digitalis, squills, and mercury to a dropsical woman; when I called to see her next day she produced with great pride a bucket full of urine which she had passed in the night. The effective remedy in dropsy cases need not, however, be a drug at all. A patient of mine with cardio-sclerosis and a regular rhythm was unable to lie down, and so sat in a chair for three weeks. Extensive dropsy super-

vened which resisted all my efforts at treatment. Finally I put the patient back to bed, and the following day he began to micturate freely and continued doing so till every sign of dropsy had disappeared.

A great many cases of heart-failure with a normal rhythm have also chronic kidney trouble; these cases are, as a rule, very resistant to treatment. Although digitalis acts here as a diuretic I rarely find any coincident effect on the heart. Still, I do not deny that there may be cases, where some special features which I have failed to note are present, in which digitalis may affect the heart. Recently I saw, on several occasions, a man of forty-six with chronic Bright's disease and a failing heart benefit greatly by digitalis. When I saw him first he had dropsy, a very rapid heart, and Cheyne-Stokes respiration, and he had had a retinal hæmorrhage; much care and treatment had been spent on him. I suggested digitalis in the manner described and it acted in a very striking manner. He was relieved of his dropsy; when the digitalis was stopped the dropsy returned. Resumption of the drug always caused the dropsy to disappear. The doctor in attendance on this patient says that the rate of the heart always went up when the digitalis was stopped and fell during its administration. I have not been able to watch the patient myself with that precision which is necessary to a clear differentiation of symptoms, and I mention his case only to show that the field for the employment of digitalis is at present far from being exhausted.

In some cases where there is a marked muscle failure digitalis acts for a time beneficially, but it is not easy to specify the particular case. When heart failure of a severe kind occurs we are of necessity forced to try all sorts of remedies. Drugs of the digitalis group offer the best prospects, and when these drugs are used they should be pushed until a definite reaction occurs, either on the heart or the digestive tract.

These drugs are often used in acute affections, as pneumonia, but in my hands I have never perceived any effect, nor have I seen the record of any positive result in the writings of others beyond the usual statement that as the patients in some cases seemed to improve after its administration, it was therefore the digitalis that caused the improvement.

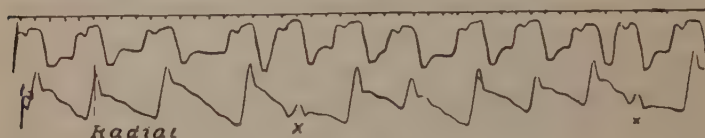


FIG. 25.

Simultaneous tracings of the jugular and radial pulses. The jugular pulse is of the ventricular type, and the tracing shows complete agreement in rhythm between the right and left ventricles. From an old rheumatic heart, in which, at the post-mortem examination, there was found great stenosis of the mitral orifice. Before digitalis. Fig. 26 was taken from the same patient.



FIG. 26.

Simultaneous tracings of the apex beat and the radial pulse from the same case as Fig. 25. The coupled beats are well marked in the apex tracing. The shading underneath shows the time of the murmurs. After digitalis. To continue the digitalis further is dangerous.

Danger in the administration of digitalis

For a long time I was at a loss to understand the warning of authorities regarding the danger of sudden death during the administration of digitalis. Of recent years, however, I have obtained an inkling into the cause of this sudden death. I have, for example, been shown tracings of the slow pulse (Figs. 25 and 26), with characteristic coupled beats, which occurs in cases of

auricular fibrillation that are under the influence of digitalis, and have been informed that the patients died suddenly. On inquiry, I found that, notwithstanding the evidences of a sufficiency, the drug was continued in large doses. I was asked once to see a man who was said to be dying of heart failure. He had to sit up in bed and he breathed heavily; his face was livid. He had dropsy, an enlarged liver, and a large and irregular heart beating at a rate of 130 to 140 per minute (auricular fibrillation.) I told his doctor to give the digitalis till he showed evidences of a sufficiency, either by the slowing of his heart or by nausea, and then to stop it. After five days the doctor telephoned to tell me that the patient was wonderfully free from distress, could lie flat, and had a good colour, that the dropsy had almost gone, and that the pulse-rate was between 70 and 80. I told him to stop the digitalis for a few days, and then, if the pulse-rate increased, to give smaller doses, and I advised him to find out the exact quantity which kept the rate about 80. Three days later he telephoned again to say that during his visit that morning the patient had fallen back and died. Until the time of death the patient had been going on well. I asked the doctor if he had stopped the digitalis as I told him and he replied in the negative, saying that, as it had done the sick man such a lot of good he had continued it, in spite of my directions.

On making inquiries regarding a few other similar cases of sudden death, I had no difficulty in recognising that the patients had been victims of auricular fibrillation, and that digitalis had been continued after it affected the heart. I myself have been following the line of treatment which consists in giving digitalis till evidences of its action are obtained, then stopping it and resuming it later, for over fifteen years. In view of the fact that I have never had a sudden death from the use of the drug, I am disposed to think that, just as there is danger in giving chloroform beyond a certain

stage, so there is danger when digitalis is pushed too far. On the other hand, if the indications I have given are followed, the catastrophe of death may be avoided.

Strophanthus.

Notwithstanding the assertion that the action of strophanthus differs from that of digitalis, I could never in practice detect any difference. Digitalis is, perhaps, more apt to produce intestinal disturbances, as nausea, than strophanthus, and so, occasionally, when there is an objection to the use of digitalis by reason of the easy production of nausea, strophanthus has been a useful substitute. I have found strophanthus produce diarrhœa very readily in a few cases. On the whole, I may repeat that, when digitalis has failed to do good in my hands, strophanthus and other drugs of the same class (squills, cactus grandiflorus) have also failed.

The tincture of strophanthus was found by my co-workers and myself to be only half as effective as digitalis. This conclusion was based upon the fact that double the quantity had to be given as a rule before a result was obtained. The tincture of strophanthus should not be prescribed in a mixture with water, as Cushny has shown that it deteriorates and after a few days becomes inert in such a mixture.

Intravenous injections of strophanthin ($\frac{1}{100}$ of a grain) act speedily, but I do not advise their use in general practice if the doctor knows how to treat his patient intelligently by the use of digitalis. Moreover, the call for the use of strophanthin injections is a very infrequent one; and when it does arise the same end which is served by intravenous injection can be accomplished in a few days by the oral administration of the drug.

Hypodermic injections of digitalin and strophanthin we found to be without any result, the drug evidently not being absorbed.

The nitrites.

The principal effect of the nitrites is a dilatation of the arterioles. It is now accepted that the cause of the dilatation is stimulation of the nerves and muscles of the vessel-walls. This effect is accompanied by an acceleration of the heart-rate and a great fall in the arterial pressure. When, for any reason, a sudden effect of this kind upon the heart is wanted, the nitrites are the most potent drugs that can be employed. A few drops (3-10 minims) of nitrite of amyl inhaled produce this effect in a few seconds. The face becomes flushed and the patient is conscious of a throbbing in the head. If the use of the drug be continued the patient becomes faint and giddy and is forced to lie down. The drug should never be continued beyond this stage. In a few minutes the effect passes off, and the blood-pressure, which has undergone a sudden fall, rises gradually and may even become higher than it was before the inhalation.

Other nitrites, such as nitroglycerin (dose $\frac{1}{100}$ to $\frac{1}{50}$ of a grain in tablets or in alcoholic solution), erythrol tetranitrate (dose $\frac{1}{2}$ to 1 grain in pills), sodium nitrite (1 to 2 grains in pills or solution), act in the same way as amyl nitrite but more slowly; the effect remains slightly longer.

Spiritus ætheris nitrosi (sweet spirits of nitre) is a very popular remedy. It contains traces of ethyl nitrite, and is often prescribed in doses of 30 to 60 minims on account of its supposed effect in relaxing the blood-vessels. As it is usually prescribed in water the nitrite which it contains evaporates rapidly, and the effect produced by it is due to ether and alcohol only.

The best effect of the nitrites is obtained in cases requiring a rapid therapeutic action; angina pectoris is an example. Some of the slower acting nitrites are recommended as reducers of a high blood-pressure, but I have had no satisfactory results from their use, nor does it seem a reasonable procedure to use them, when,

as so often happens, the increased blood-pressure may be due to some deleterious substance circulating in the blood—*e.g.*, in kidney disease.

Sedatives.

Little benefit is likely to arise from treatment so long as a patient is worried or sleepless. I have already referred to the importance of rest; it is often necessary to seek the aid of drugs to obtain rest and sleep and freedom from worry and other excitations; in these circumstances, sedative drugs, used with discrimination, are of the greatest service.

The bromides are the most useful drugs in this respect. In all cases of mild degrees of heart failure, where the patient is able to get about, but where he is worried, sleepless, irritable, or apprehensive, the bromides are extremely useful and of far more value than other cardiac drugs. I have for many years employed the bromide of ammonium, for I find that the patients are not so depressed by its use as they are when taking the bromide of potassium. The drug should be pushed until a slight lassitude is induced, or even until the patient becomes torpid, and this is true especially in severe cases of angina pectoris. The doses employed are twenty grains, two or three times a day, according to the severity of the case.

For sleeplessness the milder hypnotics—the bromides, phenacetin, veronal, and sulphonal—may first be tried. If these fail resort must be had to chloral or opium. In great restlessness, due to dyspnoea, Cheyne-Stokes respiration, or cardiac asthma, one of these drugs should be carefully given until the desired effect is obtained; five to ten grains of chloral every two hours, or $\frac{1}{4}$ grain of morphine ($\frac{1}{8}$ grain hypodermically) repeated every two hours will often suffice. When there is œdema of the lungs or bronchitis the opiates should be avoided, as they tend to check the free expulsion of the phlegm, and danger may arise from this cause. Neither chloral

nor opium should be continued for more than a few days, as the patients become sick, mentally confused, and very troublesome.

Oxygen.

The administration of oxygen in affections of the heart has been practised for many years, but the results on the whole are disappointing. Though there is certainly a limited field where the remedy seems to be of use, it is difficult to lay down precisely the conditions which call for its use. I have given it in a great variety of cases, in some with excellent results, in the majority with no appreciable benefit, or only temporary relief. Even in patients suffering from apparently similar affections the results have been unequal; for instance, a few patients suffering from cardiac asthma have been greatly relieved, while others have experienced no benefit. This difference of effects caused me to look more closely into the symptoms present, and I found that those patients who got benefit always showed evidence of cyanosis.

The conditions in which I have found oxygen occasionally of benefit are Cheyne-Stokes respiration, cardiac asthma, angina pectoris, and heart-block. Sometimes, in cases of angina and cardiac asthma, the patient has had a better night when the oxygen has been taken a quarter of an hour before going to bed. In most cases relief has occurred when the oxygen was taken during an attack of dyspnœa or pain, or, in cases of extreme heart-block, during the attack of unconsciousness.

I have followed Leonard Hill's method of administration by giving the patient large doses. Hill employs a mask which encloses the patient's head, and the oxygen is poured into the mask from the cylinder, so that the patient breathes practically pure oxygen. I have used, in many cases, a lady's hat-box, with a piece cut out for the neck. The head is enclosed in the box, the lid put on, and the oxygen given through a hole. The head

is thus surrounded by an atmosphere of almost pure oxygen. By this method, Hill states, a far greater amount of oxygen is taken up into the blood than by any other. The duration of an administration is from ten to twenty minutes. Parkinson has carried out a series of observations on the effect of oxygen given to healthy people in this way; he finds that the oxygen almost invariably reduces the rate of the heart, though only to a slight extent.

Atropine.

The principal effect of atropine upon the circulatory system is said to be due to its action in paralysing the cardiac inhibitory terminations. In practice the drug has been found to be of use in rare cases of heart-block where there has been difficulty in the passage of the stimulus from auricle to ventricle. When this state of matters exists, any increase in the difficulty may result in the complete stoppage of the stimulus. As it is well known that vagal action does produce this delay in the transmission of the stimulus, atropine, given in such cases, may cause removal of the heart-block. The drug should be given by hypodermic injection in doses of $\frac{1}{100}$ or $\frac{1}{80}$ grain of the sulphate, and repeated in half an hour if necessary.

Quinidine.

Within recent years much attention has been paid to the action on the heart of certain cinchona alkaloids, notably quinidine, an isomeric form of quinine.

This drug possesses the remarkable property of re-establishing the normal rhythm in cases of auricular fibrillation, even those of long standing.

According to Lewis this effect can be produced in about 50 per cent. of all cases so treated. This effect lasts for a varying time. Sometimes it only persists for a few days; in other cases it may be maintained for six months or a year.

The administration of quinidine is not, however, altogether devoid of risk.

Apart from personal idiosyncrasy to the drug, which has been observed in a few cases, certain untoward effects may be produced.

These are principally the inception of an extremely rapid ventricular action during its administration, and the occurrence of embolism on the resumption of the normal rhythm.

At the present time, the results of the administration of quinidine are of experimental rather than clinical importance.

Its administration must be controlled by careful and continuous observation and recording of its effects, if untoward results are to be avoided.

As this can only be carried out in a properly equipped hospital it is obviously not a suitable drug for employment as a routine therapeutic agent.

Other drugs.

A great number of agents are used in cardiac therapy, but it is doubtful if many of them have any real effect on the heart. Some undoubtedly, as, for example, alcohol and hot fluids, can show evidence of a reaction; the activity of these in producing a dilatation of the arterioles can be employed with benefit when a rapid effect is desired—*e.g.*, in attacks of faintness or prostration. It is scarcely necessary at this time of day to add a warning regarding the use of alcohol in milder forms of heart failure, and particularly in those with a neurotic tendency. The temporary benefit thus obtained may lead to a too frequent use of the drug. The danger of a habit being created cannot be overlooked.

Other drugs, such as caffeine, strychnine, and oil of camphor, act probably on the nervous system, and, by producing some exhilaration, prove useful in cases where a temporary exhaustion causes distress. But it cannot

be too strongly insisted upon that, though these remedies are often employed in cases of the most diverse kind—for instance, where there is a rapid heart in pneumonia, and where there is a sluggish ventricle in heart-block—they are without any perceptible effect on the heart and their potency is very limited. They should not be relied upon in cases of real heart exhaustion.

APPENDIX

MEDICAL RESEARCH

The defective education of the general practitioner renders him incapable of utilising his opportunities for the progress of medicine.

THIS book is intended primarily for the general practitioner of medicine. Hitherto the general practitioner has been regarded as serving a useful and, no doubt, necessary purpose in the care and treatment of those who might come under his supervision; in the larger field of the advancement of medical knowledge he is regarded, and in many cases is apt to regard himself, as a negligible quantity. So much is this so, that no attempt has ever been made to train one who intended to become a general practitioner in any branch which would enable him to undertake research work. His work, it is said, lies in a different direction. If he aspires to research work he must go to the hospitals or laboratories where facilities for such work are to be had. This is the view of medical research which dominates medicine to-day. It is legitimate to inquire to what extent this position can be maintained in the light of present-day knowledge.

To this end it is necessary to ask first, What is the nature of the problem which confronts the medical investigator? and secondly, In how far are the methods at present employed adequate for its solution?

The aim of medicine.

By common consent the ultimate object of all medical investigation is the prevention of disease. To this end it is obviously necessary to get the disease in its earliest

manifestation, when the symptoms are few, the causal conditions more easily discriminated, and the effects on the body less various than in the later phases. The disease must then be studied in relation to the conditions which favour its onset, the manner in which it invades the body, the course it pursues if left unchecked, and the measures which can be adopted to stay or influence its progress.

How the aim of medicine can be achieved.

But before we come within measurable distance of the prevention of disease, before, indeed, we can make any progress at all in this direction, one essential condition must be complied with. Not only must the disease be observed at its earliest stage, but it must be capable of recognition at the stage at which we wish to study it. It is not enough to see the disease early; we must be able to recognise it when we do see it.

This would appear to be a fairly obvious proposition, yet in spite of its obviousness it has hitherto escaped recognition. Up till now no systematic attempt has been made to investigate the processes of disease in the early stages before structural damage has been produced. With the exception of the exanthemata and a few acute infections, the early stages of disease are a closed book, yet great numbers of people are succumbing daily to diabetes, rheumatism, phthisis and a host of other diseases, the prevention of which would be of the utmost importance to the community. The hopelessness of attempting to prevent them with the imperfect knowledge of their natural history which we at present possess is only too apparent.

In spite of the mass of work which has been done, and is still being done, upon these conditions, the extraordinary fact remains that not one of them is capable of being recognised before the occurrence of structural damage and the appearance of the so-called "physical signs" of the disease. It is not, let us repeat, that

these diseases are not seen in their early stages : it is simply that they are not recognised. When they present themselves, they are apt to be regarded and treated as minor maladies, forgetful of the fact that it is to these minor maladies one must look for the beginnings of those more serious diseases which will ultimately wreck the lives of those who are affected by them.

Present methods of examination inadequate for the recognition of the early stages of disease.

Assuming then that the ultimate goal of all medical investigation is the prevention of disease, it would appear to be a logical assumption that the first step towards that end would be the recognition of disease at its inception. This raises the question as to how far the methods of investigation at present in vogue are suitable or adequate for this purpose.

At the outset we are faced by two important and indisputable facts. One is that the modern methods of clinical investigation have been in operation for a very great number of years; the other is that we are not any nearer an understanding of the early symptoms of disease than we were before these methods were adopted.

Whatever results these methods may have achieved in other directions, they do not appear to be suited to the object we have in view. A method of investigation which after years of application produces nothing cannot, by any ordinary standard, be judged efficient.

Methods of a particular kind required for the investigation of the early stages of disease.

This is not by any means to say that modern methods of investigation are valueless. All that is implied is that for the particular purpose of the investigation of the early symptoms of disease they are quite inadequate. This kind of investigation requires special methods of its own, methods which have not yet been systematically

employed, specially designed to elicit the information essential to the success of such an inquiry.

These methods are being put in practice at the Clinical Institute at St. Andrews, and are already producing useful and suggestive results. Much yet remains to be done, but sufficient has been accomplished to demonstrate clearly the applicability of the methods employed to the problem which awaits solution.

Importance of the patient's sensations.

The principal reason why the ordinary methods of examination are unavailing in the search for the early symptoms of disease is that in these stages the observer depends for his information largely upon an analysis of the patient's sensations. Mechanical methods only elicit evidence of damaged organs, but disease must be recognised before such damage occurs. The evidence of disease is present long before physical signs are perceptible. The patient is ill, and knows he is ill, long before the development of physical signs declares the true nature of his complaint.

During the time which elapses between the inception of his illness and the occurrence of physical signs the disease passes unrecognised, and, let it be borne in mind, this is the time when the great majority of patients seek advice. In other words, the patient usually seeks advice when his disease is in an early and curable stage, yet this is precisely the stage at which the examining physician is unable to say what is the matter with him.

It is of fundamental importance in the investigation of early disease that the sensations of the patient be submitted to the closest analysis. They must be studied with the same care and minuteness as would be expended on the details of a laboratory experiment.

The nature of the early signs of disease.

At the Clinical Institute it is found that the great majority of those who seek advice are not suffering

from any classical disease, but from some disabling symptom, such as an apparently trivial pain, a sense of exhaustion and incapacity, giddiness or some such sensation. Of these a certain proportion appear to recover completely, their condition being due to some temporary disturbance. Of the remainder, some improve for a time and then return with a recurrence of their disability, others, in spite of treatment, do not improve at all.

Methods of observation.

Such cases as these are continuously recorded and the development of their symptoms closely watched. In all cases the greatest attention is paid to the sensations of which the patients complain, any associated symptom is carefully noted, and the effects of rest, exercise, etc., closely observed. Further, the patient's sensations are elaborately studied while they are present, the associated symptoms which are only present when the sensation under observation is being experienced being carefully distinguished from those which occur independently of it. For instance, in the case of abnormal action of the heart, an understanding of the meaning of the patient's sensations can only be acquired by seeing patients when the abnormal action is present, by noting the changes in the heart, by auscultation, if possible by taking records by means of the polygraph or sphygmograph, and by getting the patient to describe accurately the sensations he is experiencing at the moment.

So with pain. To investigate pain properly the patient must be seen when the pain is actually present. Its position and extent must be noted, the manner in which it begins and passes off accurately defined, its character and radiation observed and the associated symptoms carefully correlated. It is essential that this be done when the attack of pain is present, for it is extraordinary how vague is the recollection of pain even of the most agonising kind. In angina pectoris, for example, when patients are asked to note the situation of pain in

future attacks, their descriptions are frequently very much at variance with the description given from memory in the first instance.

In the early stages of disease, when physical signs are conspicuous by their absence, the careful analysis of the patient's sensations acquires an additional significance. Here we must rely almost entirely upon an analysis of the patient's description of the sensations he is experiencing.

It may be argued that patients' statements are often misleading, and that dependence upon such statements will on many occasions lead to an entirely erroneous conclusion.

In some instances this is undoubtedly true, but in these instances the fault is not in the method, but in the application of it. That an erroneous result can be arrived at by the haphazard and incompetent application of a method of investigation is as true of the analysis of a patient's sensations as it is of the conduct of a laboratory experiment. Both require that knowledge and dexterity which can only be acquired by observation, practice and experience, for their successful application.

The training of the observer.

It is also essential that the patient's sensations be carefully analysed while they are actually present in order that the doctor may learn what question to ask when the patient is free from the sensation and be able to interpret his reply.

When the investigation of the early symptoms of disease was commenced at St. Andrews, it soon became apparent that the first thing necessary was for those who were to undertake the investigation to train themselves, in order that they might be able to interpret accurately the sensations of which the patient complained. Otherwise, the results attained could have no value in themselves, and there could be no effective basis of comparison between the results of different

observers. Gradually a scheme was evolved by which uniform results can be attained. The general principle underlying the scheme is that it deals with what the patient is trying to express, rather than with the terms in which he expresses himself, and reduces the sensation thus elicited as far as possible to a uniform nomenclature.

A patient's description of a sensation which he experiences varies with his individual capacity of expression. Further, it is usually coloured by his own view of its origin and nature.

It is of the utmost importance that the observer should be able to identify accurately the exact nature of the sensation or experience which the patient is attempting to describe, and to this end the methods adopted at the Institute have proved to be of the utmost value. It has been found, for example, that the common term of "palpitation" is applied by patients to indicate such various sensations as the consciousness of the heart-beat, sensations referable to gastro-intestinal peristalsis, the peculiar sensation referred to the area of the substernal notch sometimes experienced during extra-systole, and in some cases the term is used to describe the rhythmic noise in the ear sometimes heard on laying the head on the pillow at night. The same confusion is found in the use of such terms as giddiness, indigestion, numbness, tremblings—a somewhat favourite term with many patients—and many other similar complaints. These have all to be carefully and impartially analysed in order to obtain an accurate idea of what the sensation really is of which the patient is complaining.

If the practitioner will look over his own records of cases, or, failing that, the records of cases in the various Medical Journals, or even in more pretentious publications, he will frequently find such statements as that "the patient suffered from indigestion for many years," or that he "was subject to frequent attacks of palpitation," or that "he complained of nothing beyond an

occasional feeling of exhaustion and incapacity," or that "he suffered from recurrent attacks of right-sided abdominal pain"—statements in which the sensations complained of are absolutely unidentified, and consequently of very small practical value.

It cannot be too strongly insisted upon that the investigation of the early symptoms of disease is one to which the ordinary methods of investigation are, in the main, inapplicable; that the only adequate method for this purpose is to be found in the education of the observer in the careful analysis and correlation of the sensations experienced by the patient, and in a careful following up and systematic recording of the progress of each individual.

It is necessary to understand the changes effected by disease in its progress.

Turning now from the early stages of disease, we may consider an equally important subject.

It will be apparent that before it is possible to treat a patient with any prospect of success, it is necessary not only to recognise the disease from which he is suffering, but to know what is likely to happen if the disease runs its course, unmodified by any attempt to check its progress. If in its progress it gives rise to signs or symptoms, the observer must be able to detect these, and thus to appreciate the stage which the disease has reached. If there is danger to be apprehended he must know wherein the danger lies, and also whether there is any means of arresting the disease or of modifying its progress.

These are such self-evident propositions that it may seem a waste of time to detail them. But it is necessary to do so in order that the question whether we possess this knowledge may be answered. It is generally taken for granted that we do possess this knowledge; but if we inquire a little more closely we will find that such knowledge as we possess is exceedingly imperfect; and

if further we inquire into the manner in which this knowledge is to be acquired, we will speedily realise that, with the methods of investigation hitherto pursued, it is impossible that it can have been acquired.

To acquire a knowledge of the life-history of chronic diseases it is necessary to be able to follow individual cases from the start to the finish.

There is only one way of acquiring knowledge concerning the life-history of a chronic ailment. Individuals with an ailment of that kind must be observed, paradoxical as this may sound, before they become affected. The circumstances that favour the onset of disease must be recognised and the invasion of disease must be perceived. The slight varying symptoms of the earliest invasion must be detected and, as the disease progresses, the appearance of phenomena due to its presence or provoked by it must be recognised. Thus, in a disease which progresses through a period of years, the different phases, passing one into the other, must each be recognised. Consider, now, the opportunities of the profession for acquiring this knowledge. It must at once be apparent that this very necessary knowledge cannot be acquired in laboratories. The other great field of investigation—the hospital ward—cannot afford anything like sufficient opportunities, although the world has been taught to look in that direction for the advancement of medical science. The early stages of disease, before the appearance of physical signs, are rarely observed, for patients in these early stages seldom get beyond the out-patient department. Individual cases are seen only intermittently and infrequently. It may be that a large number of individuals may be seen in different stages, but all the stages are never seen in one individual, so that the merging of one stage into another escapes detection. Hence it is that our knowledge of any ailment which pursues its course for months or years is patchy and

imperfect, and does not afford a basis on which to lay a safe and sound prognosis or to build up a rational treatment.

I am perfectly well aware that this opinion will be strenuously contested, but I could illustrate my point of view from almost any chronic illness. Take, for example, the extremely common disease of the mitral valves which results in stenosis.

Here is a disease that has been universally recognised for over fifty years. It has been the subject of investigation by numerous physicians, it has been lectured upon and written about so much that there is hardly a single physician who does not imagine that all the information to be got from investigations has been acquired. Nevertheless, there is not to be found in medical literature the history of one single individual carried on from the time of the attack of rheumatic fever, which started the valve disease, up to the time of death and containing a record and due appreciation of the changes which took place.

I have endeavoured to obtain such records, but have been only partially successful. It was not until I had been engaged in research work for many years that the importance of this kind of knowledge dawned on me, and then it was too late for me to complete records with sufficient fulness. This will be understood when one reflects that such a record may demand the watching of individuals for twenty or thirty years; and as I only realised this after I had passed middle life, the reason of the shortcoming of my observations will be apparent. Moreover, at the outset I had detected many phenomena which at the time I was unable to interpret. As, however, I had made it a rule when I had perceived any manifestation with which I was unacquainted to make a note not only of its appearance but of all attendant circumstances, and particularly of the associated phenomena, the subsequent perusal of my notes, imperfect as they were, has revealed much of the

greatest importance. Had I been aware of the extraordinary light which such a record would afford at a later date I certainly would have observed my cases with greater care. I have followed quite a number of cases from the onset of rheumatic fever, and have seen the changes I have referred to gradually supervene. In the course of this work phenomena of the greatest significance were revealed, the existence of which was never suspected. Light has been thrown upon the nature and progress of disease, and incidentally certain essential features in the production of heart failure were disclosed, showing that the usual conception of heart failure in these cases—by back pressure—was misleading and erroneous. Moreover, the behaviour of the heart towards drugs of the digitalis group was shown to vary in a remarkable manner, according to the associated condition of the heart muscle. Quite a new light was thus thrown upon the nature of the actions of this group of drugs; and this new light enabled us to administer these drugs with a knowledge and precision never before attained. All these facts will be fully dealt with subsequently; here I mention them to illustrate the great limitation imposed on medical research by the imperfect opportunities of those who have hitherto instructed us in medicine, and their failure, in consequence, to obtain a thorough knowledge of the manifestations of a progressive chronic disease.

The knowledge of the progress of disease reveals the meaning of abnormal signs and constitutes the basis for an intelligent prognosis.

Not only must we investigate the progress of disease, but we must also be able to recognise the significance of signs which we assume to be abnormal. Hitherto disease in the living has been mainly recognised by the presence of abnormal signs. In a great many organs deviations from the normal are apt to occur, and it is necessary for us to understand not only the mechanism

of their production, but also their significance, so far as the future of the individual presenting them is concerned. The importance of this to the general practitioner of medicine, cannot be over-estimated.

In practice one question will arise incessantly and insistently, implied or expressly demanded by every patient, and that question is, "What is to be the outcome of my complaint?" From the point of view of the physician a diagnosis may be brilliant and productive of much satisfaction to himself: from the point of view of the patient the diagnosis is only of interest in so far as it throws light upon his future, and the problems thus presented are infinite.

Moreover, such knowledge is necessary if the patient is to be treated on rational lines. Knowledge of the condition so skilfully diagnosed must be supplemented by the knowledge whether or not the complaint is amenable to treatment, and even whether or not it requires any treatment.

It will thus be realised that what is called "prognosis" is the coping-stone which completes the edifice of one's education. All teaching should be but a preliminary training to enable one to understand this problem—the understanding of which, after all, is the most essential part of medical education. Only a mere fraction of this knowledge can be acquired during a hospital career; the fuller knowledge must be obtained from experience. It can be obtained in no other way.

It might be supposed that a matter of such vital importance as prognosis must have been the subject of careful study, and that generations of physicians must, by this time, have so thoroughly investigated the subject that the basis of a safe and sure prognosis must have been laid, at all events for the more common diseases. It is not difficult to show that this is not so.

Let me illustrate the point. In the course of his studies a student has the anatomical construction of the heart, its chambers and its valves demonstrated to

him. In his physiology he is taught the functions of the different parts. In his clinical studies he has demonstrated to him the various sounds and their modifications, and he acquires great facility in recognising the valve orifices at which different murmurs originate. He therefore flatters himself that he is in this respect qualified to enter practice. But very soon after he enters practice he may detect in some patients a murmur which his training has enabled him to recognise as arising at the mitral orifice. He may tell the patient that he or she has a murmur, and that it originates at the mitral valves. This statement may be incomprehensible to the patient, though he may be greatly impressed, but what he wants to know is what bearing this condition has upon his future, and what treatment, if any, must be undertaken. Suppose the patient is a youth; he wants to know if he is fit to play a strenuous game. Suppose he is a man; is he fit to pursue a laborious occupation? Suppose the patient is a woman; should she be allowed to become pregnant? Suppose the individual is a life insurance case; how would one estimate the probabilities of his life? When such a concrete case as this is presented to the young physician, he is at a loss. He can give a diagnosis with precision, but when the essential knowledge, from the patient's point of view, is required, he is conscious of failure. But, he may say, it is a hundred years since auscultation was introduced; the great physicians of the bygone century must have solved this question; let me turn to my text-books. He may turn to his text-books; nay, he may ransack a whole library of books, in every language under the sun, but he will get no definite answer to this simple question, "How shall I estimate the prognostic significance of this murmur?" He may inquire of authorities, men whose work it is to estimate the value of such symptoms, such as examiners for life insurance, and he will find some who will reject his patient, others who will accept him, and

others again who will increase his premium on some unknown scale; but from none will he get a clear and definite expression of the basis upon which the final opinion has been founded.

Now, if one would really understand the inward meaning of this diversity of opinion, it will be found in the fact that our methods of medical examination are developed on such lines that even experts in prognosis do not know by what means prognosis should be estimated.

How then has it come about that, with all its advances, the profession should still be ignorant of such an all-important subject? The reason is simply this, that all sciences are gradually evolved out of a stage of darkness and tradition, and medicine has not yet got so far as to be free from tradition. Certain branches are no doubt attaining the position of sciences, but such branches as treatment and prognosis are still encumbered about with much tradition and even superstition. The importance of prognosis is very imperfectly recognised. We do indeed get eloquent discourses on this subject by distinguished members of our profession; but the perusal of these discourses merely emphasises the fact that the profession has not yet realised the methods by which this branch of medicine must be pursued, nor has it recognised the one individual who is qualified to undertake its pursuit.

Investigation of prognosis can only be carried out by those who have the opportunity of watching individuals during the whole course of the disease.

To indicate clearly what prognosis is and how it should be pursued, I will cite my own experiences to show how the meaning of prognosis gradually dawned upon me. I entered general practice in 1879, and for a number of years was like a blind man groping his way. Being convinced that the wisdom of my teachers and of the authors of my text-books included all that

was to be known of disease, I felt acutely my inability to recognise obscure diseases and to tell the significance of many phenomena which presented themselves to me. Time and again my prognostications were at fault, and although I strove to remedy my ignorance by reading and visiting hospitals, I made little advance. At last, after some years, I realised that the wisdom of the teachers was not so profound and all-embracing as it appeared. I soon found that there was a bewildering variety of subjects which deserved attention. Amongst others, my attention was directed to heart affections, because this class of disease, and diseases of the digestive organs constituted the largest group of complaints among my patients. Moreover, my attention had been arrested early in my career by the case of a pregnant woman, the subject of mitral stenosis, who died undelivered, after a long and painful labour. As I had ample opportunities of studying the effects of pregnancy on the heart, I first directed my attention to this matter. As a preliminary, I studied the literature of the subject, and was surprised to find how little was known of the source of danger in heart affections associated with pregnancy. Even at this time of day, our knowledge of this subject remains very meagre. Every medical practitioner is bound to discover this very early in his career, for the problem of pregnancy and heart disease is one which sooner or later he will be called upon to face. The reason for the want of knowledge was that the profession was ignorant of the prognostic significance of almost every abnormal sign which the heart manifested. Thus I found that amongst pregnant women an irregularity in the heart's action was very frequent, and so the question arose, What is the significance—generally—of an irregular heart? On inquiring into the matter I found that there were different forms of irregularity, and so, before any advance could be made, it was necessary to recognise those different forms. At this time, over twenty-five years ago, there was no differentiation of the slightest

value based upon any reliable foundation. The first step, then—that of differentiation—had to be undertaken. Before any advance in prognosis can be made the condition with which one is dealing must be clearly defined. At the time of which I speak the first step in making a prognosis on irregular hearts had not been taken. On the careful examination of my patients I found that irregular action of the heart was the most common of all symptoms, although, in my education, the subject had never been alluded to, and in textbooks on “Heart Affections” it was barely mentioned. I had no difficulty in getting plenty of material for observation. I need not describe the long and weary steps entailed in discovering methods for recording these irregularities and in searching for a real basis on which to found a sure differentiation. Suffice it to say that, after some years, I was able clearly to recognise the character of the irregularity in the vast majority of cases and thus take the first step essential to prognosis. After I was able to differentiate the more common irregularities I put this question to myself, “What happens to those people who show these different forms of irregular action of the heart?” At the time I put this question I had no idea of the amount of labour and of the number of years that would be spent in finding out the answer. It may seem a very trivial and ordinary question and one of no particular importance. But had the question received its due recognition by the members of our profession, medicine to-day would be in a very different position. I go further, and say that very little progress will be made in medicine until those who practise it put this question to themselves and endeavour to answer it whenever they meet some sign or symptom which they do not understand. I venture to prophesy that when our physicians begin systematically to put that question, in a short time the whole attitude of the profession towards medicine will be altered.

To show what was the result of attempting to answer this question in only one of many instances, take one of the most common forms of irregular action—that due to an extra systole. This has been vaguely described as an intermittent heart. My earliest researches showed that this irregularity was, in many instances, due to a contraction of the ventricle occurring prematurely. In order to find out the bearing of the condition on the lives of my patients, I collected notes of a large number of people showing it. After a few years I had records of over a thousand cases, and from these gained information concerning the age incidence. I found that the extra systole was relatively infrequent in the young, but became more frequent after middle age, and that practically everyone over sixty years of age showed it to a greater or less degree. Then I had to watch many individuals, in order to appreciate its effect generally in relation to the stress and strain of life. Thus, pursuing my plan of investigation, I inquired what effect it had in association with pregnancy. I examined one hundred cases and found it present in fifty—in most infrequently, in a small proportion of cases very frequently. I then watched many of these women during their pregnancies. I took numerous observations when they were pregnant, and when they were not pregnant, during the period of labour when they were in pain, and when they were free from pain. Further, I kept in contact with men who pursued laborious occupations and who showed this irregularity. I watched those who were stricken down with some febrile complaint to see what effect the fever and the disease producing the fever had upon the irregular heart. I followed numbers who only showed the irregularity at varying periods and endeavoured to find out the conditions that provoked it. In some, after years of observation, I found the extra systoles became more frequent, and a few developed other irregularities—for example, that due to auricular fibrillation. Some of those who showed an extra systole drifted and died.

In some it appeared during an acute illness, and of these some died. To find out its significance in such cases necessitated an inquiry into all the other circumstances that were present, so that a knowledge might be obtained which would guide one in recognising it as a sign of grave importance or otherwise. In these and many other ways I attempted to find out the prognostic significance of this irregularity. It took years of labour and thousands of records to get the facts on which to base a prognosis. When I summarised the result of all this work I found that I had not been able to work out the subject in all its details. There are conditions (such as progressive diseases of the heart and acute affections) in which I have not been able to determine satisfactorily the significance of this sign. Thus, for instance, after an attack of rheumatic fever extra-systoles may appear. I have not been able to watch a sufficient number of cases for a long enough time to find out whether these extra-systoles are in any way connected with those fibrotic changes in the heart muscle which we find frequently after rheumatic fever, and which in many instances precede the onset of auricular fibrillation. Nevertheless, I have been able to draw certain deductions of value applicable to the majority of cases.

A misconception of how a knowledge of prognosis can be acquired has led to an inability to appreciate the results obtained by the method described.

I have from time to time published these results, and I have been interested in the manner in which they have been received. Time and again physicians of standing have stated that they were already conversant with the matter and had long recognised the significance of irregular heart action. When I have asked them why in their writings they had given no hint of this knowledge, I have been answered that the matter is one of common knowledge and needed no description. Many

practise a form of self-deception in this matter, for not only has the problem not been solved, but even the fact that such a method of investigation as I have indicated is necessary does not seem to have been realised. In text-books the prognosis of irregular action is usually simply ignored, or is referred to in some oracular utterance that can be made to bear any conceivable meaning. I was struck some years ago by an experience which revealed the disregard of this essential matter in clinical medicine. I came across a capable American who spent every fifth year in Europe studying some special subject in medicine. He had just returned from Germany, where he had been completing a period of study. He had been to a certain famous clinic, the most renowned in Germany for heart work, and had worked under a famous clinician on the heart. When he told me how he had studied and recognised the different irregularities, I asked him how he had investigated their prognostic significance. I had some difficulty in getting him to understand what I meant, so I said, "When you come across a certain irregular action of the heart in one of your patients, how are you going to estimate the bearing of the cause of that irregularity on the patient's future, and how are you going to employ it as a guide in treatment?" He seemed surprised, and said that the idea had never occurred to him and had formed no part of his study. After I pointed it out he realised its real importance to him as a general practitioner. He came to Burnley, where I was practising, and stayed there three or four days. I showed him a great many patients with irregular hearts, with the notes of their past histories. Some of these patients were leading strenuous lives, others led but a crippled existence, and I was able to show him the indications that would guide one towards a prognosis and a rational treatment. After his return to America he began to apply his knowledge, and he wrote me to the effect that he had now obtained an

insight into the meaning of the subject and to a knowledge which he could apply intelligently in practice, and that, so far as this knowledge was concerned, he had learnt more in three days in Burnley than he had in six months in Germany.

Not very long ago I visited a famous Continental clinic and was presented by a young physician, who was in charge of the cardiac department, with a work he had written on the extra-systole. In this work the subject was dealt with after the conventional manner, under different headings, including prognosis. I said to the author, "I see you deal with prognosis. Have you settled the matter?" "Oh, yes," he replied. I then inquired during how long a period he had been able to recognise an extra-systole, and found that he had been able to recognise it during two years. His observations had been confined to the bounds of the hospital. So it would seem that this young man thought himself capable of deciding in two years a matter I had spent twenty-five years upon and which I had not been able to complete.

I use this illustration not in any way to reflect upon the eager young men who are to-day making the same mistake—I have myself made many foolish mistakes of the same sort—but to show that the profession has not yet awakened to a realisation of what prognosis really means. Every member of our profession is labouring under the disadvantages inseparable from an imperfect idea of what constitutes prognosis. How severely the profession is handicapped by this lack of appreciating the true method of investigation is seen in almost every phase of clinical medicine. No sooner is a new method introduced than its users begin to employ it as a means of prognosis, long before the nature of the information it reveals is understood. Take, as an illustration, the attitude of the profession towards murmurs. Within a few years of the discovery of auscultation as a method of diagnosis—a hundred years ago—it was observed that

murmurs were found in patients with heart trouble who subsequently died of their disease. The physician of that day, not knowing what was the cause of the murmur, attached a grave significance to its presence. To-day the whole profession is obsessed by these early observations. Physicians who are careful observers have been able to get a fair idea of the significance of different murmurs, but even then the knowledge is not so precise and definite as to enable them to show the reasons for their beliefs. We can see to-day the same process at work in the case of blood-pressure instruments as in the case of the stethoscope. We still find many physicians solemnly writing of the significance of blood-pressure, while as yet we are ignorant both of what the term is intended to convey, and of what the normal pressure may be. If anyone cares to test this statement, let him look up the literature on this subject written during the last ten or fifteen years.

In thus drawing attention to the imperfect knowledge of what constitutes the science of prognosis, it must not be thought that I am describing faults from which I myself am free. Far from it. I was reared and bound in the bonds of tradition, and have indulged in all these faults and hasty conclusions. It was only after weary years of study and finding myself so often at fault that I gradually realised the significance of that question which I had put: "What bearing has the cause of this abnormal sign on the patient's future?"

The opportunity for investigation in hospitals is too restricted.

If one wishes to gain an idea of the reason why medical science advances so slowly and haltingly one has but to look at those who are at present the investigators and to consider their opportunities. To prevent or combat disease it is necessary to know its origin and progress; and one has but to ask what opportunities our investigators have of seeing the disease invade the

body, and of observing its progress, in order to estimate the amount of knowledge likely to be acquired by them. Investigation is chiefly carried on in the laboratories and in the wards of hospitals. No doubt in both places observations of the utmost importance are made; but what opportunities have these investigators of seeing disease in its early stages and during its progress? In the wards of the hospital, as we have seen, disease is not presented in its early stages, and the opportunity of watching its progress is limited to the short time the patient spends in the hospital. It is true that in the out-patient department many cases of disease in its early stages may present themselves, but here again the difficulty of following cases is well-nigh insuperable. In addition, the opportunities which do present themselves have been missed. Bearing in mind the necessity of appreciating the sensations of the patient, and the fact that it is only after long years of study and preparation that the knowledge as to how to elicit these sensations can be acquired, it will be readily seen how useless it is to expect any advance from the out-patient department as it is at present administered. I called attention to this some time ago, pointing out that, when disease is in its earliest and most curable stage, and consequently most difficult of detection, patients are sent to the out-patient department and left to the least experienced members of the hospital staff. On the other hand, when the disease is most advanced and often actually incurable, and the signs most easily perceived, the patients are admitted to the wards and placed under the care of the senior and most experienced physicians. Patients whose symptoms are purely subjective present themselves at the out-patient department; and, the nature of their complaints not being recognised, they are put off with a bottle of medicine and told to return if no better. They keep returning until the disease is so far developed as to show some physical sign, after which, but not before,

they are considered fit for admission into the hospital. Then they obtain all the resources of the ward and laboratory investigators.

The general practitioner is the only investigator that has the real opportunity.

But it is only a very small proportion of the sick who visit a hospital. The vast majority of persons, when they first perceive that there is something amiss, turn to their family doctor and continue under his care. It is the general practitioner, who sees disease in its early stages, who has the opportunity of finding out what conditions predispose to the disease and of following it through all its stages.

If one reflects upon the nature of the knowledge that is essential to medical research, and then upon the opportunity of the men who devote their time to research, one is forced to the conclusion that this opportunity is greatly restricted. And, further, the conclusion is inevitable that only one type of investigator can carry out this particular, but essential, line of research, namely, the general practitioner. I am not deprecating the work of the hospital or of the laboratory—all are required. I am only pointing out why progress is so halting, and showing that it is bound to be fettered and restricted until the man with the opportunities takes his place as an investigator. If the signs of the past had been read aright this would have been recognised long ago. If anyone cares to inquire how the efficacy of vaccination was discovered, he will at once realise that Jenner might have spent his lifetime in laboratories and hospital wards and yet never have made his discovery, simply because he would never have had the opportunity.

This need for the general practitioner as investigator, which will, in time, become self-evident, is, at the present day, not realised. We see money poured out for the advance of medical research, but no one thinks

of utilising the opportunities of the general practitioner. Some endowments are specially guarded, indeed, as if to prevent the general practitioner from participating in them. The domination of the laboratory is so great that the whole tendency is for medicine to be ruled by the conception of the laboratory worker. So much is this so that it is supposed to be a sign of progress when a teacher of clinical medicine is appointed who has received his training in a hospital or laboratory. The method of selecting as teachers of medicine men who can have no knowledge of the aspects in which disease will present itself to the vast majority of their pupils is utterly indefensible. There is a vast difference between hospital practice and general practice. Such teachers, with their limited training, are practically unconscious of the great opportunities for the advancement of medicine which their pupils who go into general practice will have. If these salient facts are fully grasped it will be realised that what is called progress in teaching—*i.e.*, leaving teaching exclusively in the hands of laboratory-trained men—is but the forging of fetters to bind the one class of men whose help is essential to the progress of medicine.

A new spirit is essential to medical research.

If progress is to be made, a new spirit must be infused into teaching and into research. If the most enlightened members of our profession were to inquire into the grounds of their beliefs, even those most dearly cherished, how often would they be surprised to find the fallacies on which their beliefs are based. Medicine is being evolved out of ages of tradition, and the bonds of tradition are difficult to shake off. It is not only the superstitious beliefs of bygone ages that hamper us, but also the spirit of tradition, which is ever present; for the tendency to believe without reason is ever recurring, while the reverence for authority makes us accept statements without proof.

I could illustrate this in almost every field of medicine, but the most notorious example is to be found in our methods of treatment. Take the official pharmacopœia and inquire into the grounds on which the various drugs have gained admission to its pages. The vast bulk of these drugs have never been subjected to any accurate observation, and many have gained admittance because the popular belief attributed to them some supposed effect—evidence that would equally justify the inclusion in the pharmacopœia of the King's touch, amulets, and invocations. Even the few drugs that are of value have never been observed with that precision which would enable the student to understand how to make the best use of them. I trust it will not be long before the most recent of pharmacopœias will be regarded as bearing the same relation to therapeutics as a mediæval work on alchemy does to modern chemistry.

The inquiring spirit, in seeking diligently for true methods of acquiring knowledge and for obtaining subjects for investigation, cannot follow a better way than to ask, What is the basis of my belief? No doubt, in our early days, we must be content with the statement of authority. But those whose duty it is to teach should constantly be inquiring into the reasons for the beliefs they seek to inculcate into their students. If this were done systematically not only would the teacher be surprised at the nature of his own reasons, but the very recognition of the baselessness of his beliefs would stimulate him to inquire. And once the inquiring spirit was aroused the good it would accomplish need not be insisted upon nor the lines it would pursue be a matter for anxiety.

My purpose in insisting upon the necessity for study of the patient's sensations, and for the utilising of the opportunities of the general practitioner, is to demonstrate that, in clinical medicine, methods of research must be adopted that are peculiar to this branch of

science. It is repeatedly stated by leading members of our profession that the limits of clinical medicine have been reached, and that no further progress can be made until new methods have been discovered. This belief is widespread, and is seen in the sudden rush which is made after any new mechanical device. I want to insist upon the fact that the means of investigation which we possess in ourselves, without the use of mechanical devices, have not yet even been begun to be employed on a scientific basis.

Exact methods are now widely recognised as necessary in investigations, and as their recognition is the outcome of laboratory practice, laboratory methods have at present a dominating influence in medical research. But as yet we scarcely perceive the limitations of laboratory methods. In the discovery of the mechanism of some obscure process laboratory experiments are essential; but to find out what bearing that obscure process has upon a patient's future life is quite another matter. Nevertheless, it is an essential matter. It follows, then, that research in clinical medicine must pursue lines of its own; these lines are not the loose, speculative ones of bygone days, but the precise and accurate observations on living individuals by means of which alone knowledge of real value can be obtained.

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